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THE REPAIR OF HIATUS HERNIA OF THE DIAPHRAGM BY THE SUPRADIAPHRAGMATIC APPROACH*

Technic and Results

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THE use of the supradiaphragmatic approach in the treatment of esophageal hiatus hernia converts what, in the majority of cases, would be a difficult and formidable procedure into one that can be accomplished with ease and simplicity. It eliminates all the difficulties of technic experienced when the operation is done from below the diaphragm. Handling of any of the abdominal viscera except those within the hernia sac is avoided; the exposure to the operative field is wide and direct; the freeing of the sac is readily accomplished; and structures that are to be sutured are easily seen and dealt with. This approach also makes it easy, in cases in which the esophagus is short, to overcome the difficulty mentioned by Harrington[1] of obtaining fixation of the esophagus to the edges of the reconstructed hiatus. To repair a hiatus hernia of the diaphragm through the abdomen is analogous to attempting to repair a large inguinal hernia through a laparotomy incision, working at the opening of the sac from within.

Further important advantages of the use of the thoracic approach in the repair of hiatus hernia are the relative freedom from postoperative complications and particularly the much lower incidence of recurrence of the hernia after the operation.

ANATOMIC FEATURES OF HIATUS HERNIA

That a hernia through the esophageal hiatus of the diaphragm is a true hernia is proved by the fact that in all cases, excluding traumatic rupture of the diaphragm extending through the hiatus, there is a peritoneal sac. But unlike that in inguinal hernias, the wall of the sac never consists entirely of free peritoneum. All cases of hiatus hernia are, in fact, of the sliding type involving the wall of the fundus of the stomach, which makes up one surface of the sac in much the same way as the wall of the cecum or of the sigmoid becomes a component of the sac in certain cases of inguinal hernia. An understanding of this anatomic relation of the structures involved helps to explain what must be accomplished to relieve the pain induced by these hernias and is necessary to perform a repair that will not result in a recurrence.

These hernias can be classified according to the changes that result in the relations of the esophagus and stomach at the level of the hiatus. In the majority of cases, the stomach bulges upward through the hiatus, pushing the esophagus up or at least allowing it to retract. This anatomic change in the relation of the viscera makes it appear as though the esophagus were abnormally short. The failure to understand the true nature of this condition explains the frequent recurrences that make the use of the abdominal approach so disappointing to the average surgeon.

Although a true congenital shortening of the esophagus is occasionally observed in cases of hiatus hernia, it is fortunately very unusual. In such cases the cardia cannot be drawn down to a point at or below the level of the hiatus by any means. The condition therefore remains incurable from the purely anatomic standpoint, although if the peritoneal sac is ablated, a cure of the symptoms usually results.

In a few of the cases, the hernia is of the so-called paraesophageal type. The esophagus maintains its normal length and position, but the stomach along with the herniated peritoneal sac passes upward between the margin of the hiatus and the esophagus. This type of hernia, though relatively infrequent, is responsible for some of the largest herniations but is fortunately more easily corrected than the other.

The esophageal hiatus is, of course, the defect through which the hernia passes. In the majority of cases, regardless of the type, the difficulty arises from a simple enlargement of this opening, probably on the basis of a congenital weakness without absence of muscle or other defect in development at any point around its circumference. The shape of the enlarged hiatus varies somewhat from case to

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case, but in the majority it is ovoid rather than round. This fact favors the placing of the sutures used to produce a narrowing of the opening. Also, in the majority of cases the direction of this ovoid enlargement of the hiatus is transverse or oblique.

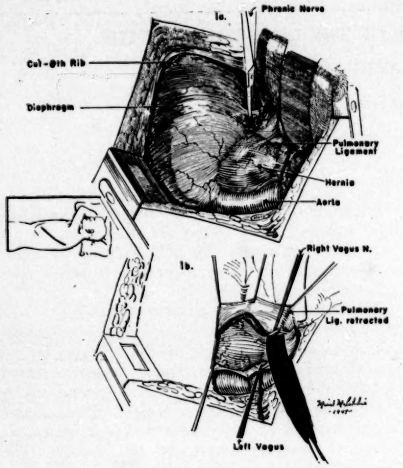

FIGURE 1. A *Shows the Exposure of the Operative Field Obtained by Incision through the Left Hemithorax, Excising the Eighth Rib, and* B *Dissection of the Hernia, the Lower Esophagus and the Margins of the Esophageal Hiatus after Incision of the Mediastinal Pleura. (Inset: Direction of the Incision on the Chest Wall.)*

Rarely is it anteroposterior. This fact must likewise be borne in mind when sutures are being placed.

In rare cases there is a congenital absence of a portion of the diaphragm, which should normally make up a part of the margin of the hiatus. The correction of this condition presents technical difficulties that offer further opportunity to demonstrate the superiority of the thoracic approach. It is highly improbable that such a defect could be corrected through an abdominal incision.

Description of the Operation

With the patient lying on the right side and the left arm drawn up in front of his face, a long oblique incision is made across the left side of the chest in the direction of the ribs. Because of the fact that the majority of patients with hiatus hernia who require surgery are of middle age or older and therefore have an inflexible chest wall, it is better to resect a rib than to attempt to obtain sufficient exposure by an intercostal incision. Resection of the eighth rib gives the best results in the average case. A rib spreader is inserted, and the lower lobe of the lung is retracted. The exposure obtained by this means is illustrated in Figure 1. The hernia sac and contents are easily identified lying beneath the pulmonary ligament and the pleural reflection over the lower mediastinum (Fig. 1*A*).

The left phrenic nerve is crushed with a hemostatic forceps at the point where it leaves the surface of the pericardium to enter the diaphragm. The tranquillity of the diaphragm thus induced not only assists in the performance of the operation itself but also, by ensuring temporary inactivity of the muscle, encourages firm healing of the structures that have been sutured. When the abdominal approach is used, this step must be performed through an incision in the neck, as Harrington[1] advocates.

The mediastinal pleura overlying the hernia is incised longitudinally and removed from its attachment around the margin of the hiatus. The hernia

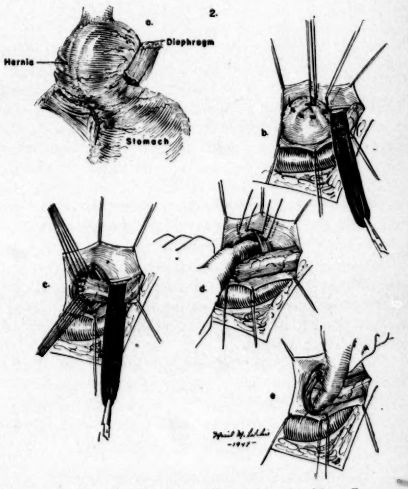

FIGURE 2. A *Is a Diagram Illustrating the Most Frequent Type of Hernia, with an Apparent Shortening of the Esophagus Brought about by the Upward Thrust of the Herniated Portion of the Stomach, the Sac Consisting of a Reduplication of Peritoneum over the Anterior Aspect of the Herniated Stomach.* B *Demonstrates the Beginning of the Repair, Showing Plication Sutures Used to Infold the Peritoneal Sac.* C *Shows Complete Reduction of the Hernia Produced by Obliteration of the Sac, with Plication Sutures Already Tied but not yet Cut.* D *and* E *Illustrate Placement of the Sutures Used to Narrow the Esophageal Hiatus.*

sac, the herniated portion of stomach, the lower end of the esophagus and the overlying vagus nerves are freed by dissection. The margins of the hiatus are identified and cleared of the areolar tissues that are found attached to them (Fig. 1*B*).

The ultimate success of the repair depends upon the observance of two important steps in the technic. In the first place, the hernia sac must be eliminated. This is accomplished in the majority of cases by means of a series of plicating sutures of silk placed in the free peritoneal portion of the sac in a circumferential direction (Fig. 2*B* and 2*C*). Before this layer is inserted, it is often possible to free much of the sac from its attachment to the underlying wall of the stomach near the cardia. This serves to make plication of the sac easy, but is not essential to the success of the procedure. It is not necessary to open or to remove the sac in the average case.

In a few cases, however, it will be found that the enlargement of the peritoneal reflection that forms the major portion of the wall of the hernia sac has developed far beyond the extent of the stomach into the chest. In such cases a large redundant portion of the sac can be freed and removed. The base of this type of sac must, of course, be closed either by a suture ligature or by means of a series of mattress sutures. The remainder of the sac is then plicated in the usual way.

Occasionally, when the sac is a large one, it is necessary to insert two layers of plication sutures to maintain a reduction of the hernia. If the sac

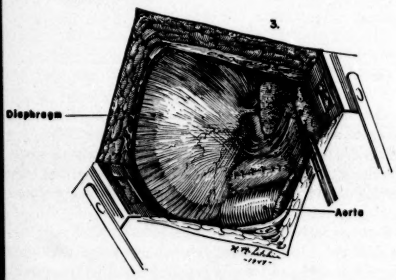

FIGURE 3. *View of the Left Hemithorax before Closure of the Chest-Wall Incision.*

Suture of the opening in the mediastinal pleura is shown after completion of the repair.

has been properly inverted by plication or removed, the hernia remains reduced with the cardia at or just below the level of the hiatus (Fig. 2*C*).

The second important step of the operation is to narrow the hiatus to such a size that the reduction of the hernia can be held permanently. This is accomplished by means of several sutures of heavy silk placed solidly through the muscle well behind each edge of the hiatus. Because of the natural transverse direction of the opening in the majority of cases, these sutures must be placed at each side of the esophagus. It is usually necessary, because of the relation of the esophagus to the opening, to put more of these sutures to the left than to the right (Fig. 2*D* and 2*E*). One layer usually suffices, but two layers may be necessary if the diaphragm is unusually thick. It is unwise, however, to apply a second layer if it produces any tendency to roll the edge of the diaphragm up onto the esophagus, because of the danger of causing an angulation just above the gastric cardia. The repair can be given greater strength and permanency by the insertion

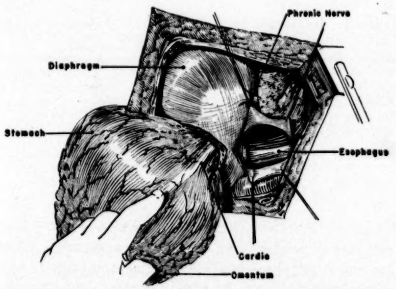

FIGURE 4. *Diagram Illustrating the Method Suggested in Cases in Which the Enormous Size of the Hernia Makes It Difficult to Maintain Reduction While the Repair Is Being Carried Out. The stomach and omentum have been pulled out through a counterincision in the diaphragm. After repair of the hernia has been completed, the stomach and omentum will be replaced in the abdomen, and the opening in the diaphragm closed.*

of a reinforcing suture of fascia lata obtained by an assistant from the patient's left thigh.

In narrowing the hiatus of the diaphragm it is necessary to exert great care to avoid a constriction that might lead to delay in the passage of food through the esophagus at that level. A good rule to follow is that if the index finger of the surgeon can easily be inserted beside the esophagus, the correct size of the opening has been established. By adherence to this rule, postoperative dysphagia has been avoided in all cases.

To complete the operation, the mediastinal pleural incision is closed with fine-silk sutures, and the edges of the pleura reattached to the diaphragm near the hiatus (Fig. 3). The lung is then expanded by the anesthetist, and the chest-wall incision is closed in the usual fashion, interrupted silk sutures being used.

Difficulties Encountered

In certain cases in which the hernia is unusually large, consisting of almost the entire stomach, a large part of the transverse colon, the great omentum and sometimes a portion of the small intestine as well, it is impossible to maintain a state of reduction while the repair is being made. In some of these cases, as in certain enormous scrotal hernias,

there seems to be insufficient room within the abdomen for the long-displaced viscera. To overcome this difficulty it is necessary only to make a short counterincision in the diaphragm well away from the hiatus. Through this incision the herniated viscera are easily pulled into the abdomen and if necessary out over the lower edge of the thoracic incision, where they are protected with a pad of wet gauze (Fig. 4). The sac can then be dealt with, and the opening in the hiatus reduced to proper size with less difficulty than in the average case. After the repair of the hernia has been completed, the viscera are replaced in the abdomen and the incision in the diaphragm is closed. If the sutures used to close this incision tend to pull through the tissues because of the increased pressure within the abdomen, a strip of fascia lata will overcome the difficulty.

It sometimes happens, when sutures are placed across the hiatus to the right of the esophagus, that the branch of the right vagus nerve that passes to the celiac ganglion interferes. In such a case, this branch of the nerve should be divided. No untoward result has been observed to follow this maneuver in the few cases in which it has been necessary.

If the esophagus is actually too short and a portion of the stomach is therefore congenitally thoracically placed, no attempt to change the situation should be made. Furthermore, it is not necessary in such cases to elevate the diaphragm in an attempt to relieve the patient. The pain is eliminated if the peritoneal sac is removed, after it has been dissected free from the stomach, or if it is thoroughly inverted below the level of the hiatus.

TABLE 1. *Indications for Operation in 51 Patients Operated upon for Hiatus Hernia.*

INDICATION	NO. OF CASES	PERCENTAGE
Intractable pain	35	68.6
Blood loss	7	13.7
Chronic	5	9.8
Acute	2	3.9
Incarceration with or without obstruction	9	17.6

TABLE 2. *Complications Following Supradiaphragmatic Repair of Hiatus Hernia of the Diaphragm (51 Patients).*

COMPLICATION	NO OF CASES
Empyema	1
Thrombophlebitis of leg veins	3
Sublethal pulmonary embolism	1
Minor wound sepsis	1
Total	6*

*Five patients.

POSTOPERATIVE CARE

The management of the early convalescence of the patient offers no special problems other than those likely to be encountered whenever a thoracotomy is performed. It is wise to leave a Levin tube in the stomach during the first forty-eight hours to prevent gastric dilatation. The patient should be fed with caution at first, but a normal diet is usually tolerated by the seventh or eighth day. Early ambulation is encouraged. The majority of patients are out of bed from the second day on. The average period of hospital stay after the operation is twelve and a quarter days.

CLINICAL EXPERIENCE

The operation described above has been used in the treatment of 51 patients with hiatus hernia of the diaphragm. In every case an important indication for the operation existed. In the majority of cases the reason for operation was the occurrence of severe attacks of pain, which could not be controlled by dietary or medicinal means. In a few cases a severe degree of blood loss, usually of a chronic nature, provided the indication for surgery. In the remainder there was incarceration of the hernia, usually with obstruction, either intermittent or complete (Table 1).

TABLE 3. *Late Results of Supradiaphragmatic Repair in 43 Patients Operated upon Six Months or More Ago.*

RESULT	NO. OF CASES
Incisional discomfort:	
Severe	0
Slight	4
None	36
Not stated	3

Postoperative complications occurred in 5 patients (Table 2). One patient, whose operation was performed early in the series before penicillin was available, developed a localized empyema, which required drainage. Thrombophlebitis of the leg veins developed in 3 cases, in 1 of which a sublethal pulmonary embolism occurred. The only other complication consisted of minor, superficial wound infection, which was not serious enough to delay discharge from the hospital.

In a comparable series of 25 cases of hiatus hernia repaired at the Massachusetts General Hospital by other surgeons using the abdominal approach, there were complications in 7 cases and 1 postoperative fatality.

There were no postoperative deaths among the 51 patients operated upon transthoracically.

For the information of those who may hesitate to use the thoracic approach because of the fear that the incision may remain painful for a long period after the operation, a careful inquiry was made of

all the 43 patients whose operations were performed six months or more ago. A definite statement was obtained from 42 of these patients. In no case had there been any severe pain after the immediate postoperative period. Four of the patients reported slight discomfort, but 38 patients experienced no discomfort from the incision after the first few weeks (Table 3).

Results

Eight patients have been operated upon so recently that a follow-up study of the result would hardly be significant. The remaining 43 patients, however, were operated upon six months or more ago, and the results of a recent study of their present status are illustrated in Table 4. Criteria for evaluation of the end result in these cases have been based upon the statements of the patient and his physician regarding the relief of symptoms and, whenever possible, upon roentgenographic evidence obtained by the ingestion of barium. In none of these cases has a definite recurrence of the hernia been demonstrated by roentgen-ray studies. In 1 patient there is evidence still of a slight protrusion of the cardiac end of the stomach above the hiatus. In this case, however, the reduction of the herniated stomach was incomplete at operation because of an abnormally short esophagus. Furthermore, the patient, who was a chronic invalid because of the severity of the pain before operation, is now cured of the pain caused by the hernia. He continues to have occasional attacks of angina pectoris from known pre-existing coronary-artery disease. In another patient there is an equivocal shadow in a roentgen-ray film obtained several months after operation. This case is classified as suggestive of recurrence although the symptoms were completely relieved by the operation.

The failure to relieve the discomfort in 1 case may mean that the symptoms were erroneously ascribed to the hernia. At any rate, no roentgen-ray evidence of recurrence has been observed in this patient. The same thing may be said for the 3 patients who are only partially relieved of discomfort. In all the other 39 patients, including those who had bled and those with an incarceration of the hernia, as well as those who had pain only, the relief of symptoms has been complete and permanent (Table 4).

Soutter[2] found after a review of 24 cases of hiatus hernia repaired through the abdomen at the Massachusetts General Hospital that a large number had apparently recurred, often before the patient left the hospital. In the 9 patients of this group who had subsequent investigation by roentgen-ray studies a definite recurrence was observed. On the other hand, of the 43 patients operated upon by the thoracic approach six months or more ago, 95.5 per cent show no suggestion or evidence of recurrence. This result, when compared with the results obtained by the use of the abdominal approach, at least among the cases observed at the Massachusetts General Hospital, demonstrates the superiority of the supradiaphragmatic operation.

Table 4. *Late Results of Supradiaphragmatic Repair in 43 Patients Operated upon Six Months or More Ago.*

Result	No. of Cases
Evidence of recurrence:	
Definite	0
Suggestive	1
Hernia not reduced completely at operation	1
None	41
Symptomatic relief:	
Complete	39
Partial	3
None	1

Conclusions

The widely prevalent belief among physicians and some surgeons that an operation for the repair of hiatus hernia of the diaphragm, especially when performed by the thoracic approach, is unusually dangerous or attended by a high incidence of complications is erroneous.

The widespread impression among physicians and surgeons that the repair of a hiatus hernia of the diaphragm is likely to be followed by a high incidence of recurrence is based upon experience with the abdominal approach and does not apply when the supradiaphragmatic operation is used.

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PROLONGED PAROXYSMAL AURICULAR TACHYCARDIA

Report of a Case Including Treatment with Veratrum Viride

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THERE have been several reports in the literature of cases of prolonged paroxysmal auricular tachycardia. The case reported below is presented because it is somewhat rare and it is the first case in which veratrum viride has successfully though temporarily stopped an episode of tachycardia.

Peterman[1] reported a quite similar case in a six-year-old child who developed an auricular rate of 160 in November, 1943, and at the time of the study (January, 1946) the tachycardia had persisted. Digitalis was given until toxic effects were produced without stopping the tachycardia; mecholyl produced only temporary conversion to normal rhythm, and papaverine, ipecac, quinidine and intravenous magnesium sulfate were without effect. An inflated blood-pressure cuff about the neck caused an immediate conversion to normal sinus rhythm, but the paroxysmal auricular tachycardia returned the next day. There was no known cause of the tachycardia, though it was first noted after several very severe upper respiratory infections.

Mayer[2] described a case in a ten-year-old girl who had had a persistent tachycardia starting at the age of one. There was no known precipitating cause.

Piotti[3] reported a case in a child first observed at the age of eleven months in an episode lasting twenty-three days. A later attack lasted seven months. Various therapeutic agents were tried, including digitalis, quinidine and ergotamine tartrate, none of which had any effect. The patient finally died in congestive heart failure at the age of eighteen months. Histologic examination of the heart at autopsy revealed an interstitial myocarditis of the Fiedler type. The lesion was located only in the right auricle and in the region where the fibers of connection run from the ostium of the coronary sinus to the node of Aschoff Tawara.

Mahaim[4] reported a case very similar to that of Piotti, in a fourteen-month-old child who developed the condition after a respiratory infection. The initial paroxysm lasted fifteen days and was followed by another during which the child died of bronchopneumonia. Post-mortem examination revealed some recent inflammatory lesions of the Keith–Flack node.

Cooke and White[5] reported an episode in an infant aged eighteen months that lasted over twelve months without any apparent ill effects. This was the only case of long duration in their series of 600 cases.

Olney[6] has seen several cases of prolonged paroxysmal auricular tachycardia in children, usually after infectious diseases. She states that the cases are usually benign and terminate spontaneously and are better left untreated if the heart is otherwise normal.

In adults, Hamilton and Hurwitz,[7] Herson and Willington,[8] Maddox[9] and Miller and Perelman[10] have reported cases. In reviews of large series of cases Gallavardin[11] found that 11 out of 160 patients had attacks lasting as long as ten days; 4 of these cases lasted over a month. None of the patients died during the attacks. Campbell[12] found 2 cases in his series of 100 that lasted longer than ten days. Hume[13] reported that only 1 of a series of 48 cases lasted over ten days. Levine[14] has followed a patient who had paroxysmal auricular tachycardia for twenty years; the auricular rate persisted at 240, and the ventricular rate was usually 120 but occasionally 60 or 240. Bouveret,[15] in the classic early article on paroxysmal auricular tachycardia, described a case lasting thirteen days.

CASE REPORT

J. C., a 3-year-old boy, was first seen in the South Department of the Boston City Hospital for an acute pharyngitis and otitis. He received symptomatic treatment with prompt recovery. The heart rate and rhythm were normal at that time. He was next seen at the age of 5 on February 4, 1947, with a history of nasal discharge for 2 days and sore throat for 1 day.

There was no past history of any joint pains or palpitation. The family history was noncontributory, with no history of heart disease. The mother was 24 and the father 27 years of age; both were living and well. Four younger siblings were also living and well.

Physical examination disclosed a well developed and well nourished boy. There was a tonsillar membrane. The remainder of the examination was negative. The heart was not enlarged, the sounds were of good quality without murmurs, and the rate was 90 with normal rhythm.

The patient was given 30,000 units of diphtheria antitoxin intramuscularly. The nose and throat cultures proved positive for diphtheria bacilli. The membrane rapidly disappeared, and convalescence was uneventful. The patient received penicillin parenterally from March 11 to March 20 because of continued positive diphtheria cultures from the throat.

On March 18 the patient developed varicella. On April 3 the tachycardia was first noted. An electrocardiogram revealed a regular rate of 140 with inverted P waves in the limb leads. The pulse was regular, but the rate increased with emotion or exertion; it was unaltered by carotid-sinus or eyeball pressure.

On April 11 the patient developed a moderate case of measles, without complications. Two days later an electrocardiogram revealed a rate of 94 with normal upright P waves except in Lead 4_R, in which, after a deep breath, the rhythm changed from a paroxysmal tachycardia with upright P waves to a sinus rhythm with inverted P waves. Earlier that day and after the electrocardiogram the pulse had been 130 and regular.

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On April 21 quinidine sulfate was given. Some irregularity of the pulse was noted after the third dose of 0.2 gm. An electrocardiogram revealed occasional runs of sinus rhythm interspersed in the paroxysmal auricular tachycardia. On the following day quinidine was given up to 1.2 gm. without effect.

On April 23 digitoxin was started orally. Toxic symptoms were reached, and runs of normal sinus rhythm occurred but there was no prolonged conversion. About 3.5 mg. was given in 4 days. On May 1 the patient was discharged and allowed full activity at home.

On May 12 0.2 gm. of quinidine was given three times daily in addition to 1 teaspoonful of potassium acetate in 25 per cent solution three times daily. Because of vomiting of the potassium acetate potassium chloride solution was tried for 4 days, but without effect.

From May 23 to June 20 the patient was given 0.1 mg. of digitoxin daily and 0.2 gm. of quinidine three times daily, only to become toxic again without converting to sinus rhythm.

On June 24 the patient re-entered the hospital for esophageal electrocardiograms and a trial of mecholyl. Large doses of phenobarbital (up to 0.15 gm. three times daily) over a week were first given in an attempt to obtain sedation. This therapy effected no appreciable degree of sedation, nor did it alter the tachycardia. Sodium pentothal anesthesia, 1.2 gm. by rectum, was given on June 26 with prompt and effective anesthesia, which enabled performance of esophageal-lead electrocardiograms. On July 3 mecholyl was given for the first time, the technic described by Starr[16] being used. With the patient lying horizontal, alternate pressure over the carotid sinuses was tried; when this was fruitless mecholyl was administered. The first dosage was 1.25 mg. When the tachycardia persisted after ½ hour a larger dose (2.5 mg.) was given. A third dose of 5 mg. produced facial flush and sweating about 3 minutes after subcutaneous injection. The rhythm changed to a sinus tachycardia rate of 160, with

FIGURE 1. *Electrocardiogram before the Administration of Vertavis, Showing Paroxysmal Auricular Tachycardia in All Three Limb Leads.*

upright P waves in Leads 1, 2 and 3, then to a shifting pacemaker for a minute and then back to the paroxysmal auricular tachycardia. On July 7 the patient was given 6.25 mg. of mecholyl subcutaneously; about 2 minutes later the rhythm switched from the tachycardia to a sinus rhythm at a rate of 80. He sweated, flushed, passed flatus, became nauseated and complained of abdominal pain; 0.0006 gm. of atropine was given subcutaneously, with prompt cessation of the side effects. The normal rhythm lasted about 10 minutes, and then the tachycardia recurred.

The patient was next given a trial of veratrum viride. The first tablet of vertavis* (10 Craw units) was given on July 22 at 4:35 p.m., when an electrocardiogram disclosed paroxysmal auricular tachycardia in all three limb leads (Fig. 1). The pulse was checked every 10 minutes, and the blood pressure

*Kindly furnished by Irwin, Neisler and Company, Decatur, Illinois.

every ½ hour. At 7:25 p.m. the pulse rate started to fall, from the preceding 136 to 114. By 8 p.m. the rate had reached 80 and the electrocardiogram revealed normal sinus rhythm with upright P waves (Fig. 2). The pulse rate remained below 100 until 10 o'clock the next morning, when it started to rise. By 3:30 p.m. the pulse was 140, with the usual inverted P waves in the electrocardiogram. There was no effect on the blood pressure, which always remained around 110/70, nor were there any subjective symptoms. On July 23 at 4:30 p.m. the patient was given one tablet of vertavis. Five hours later the pulse had fallen to 92, and the electrocardiogram showed upright P waves. At 7 o'clock the next morning the pulse rate had reverted to 140. The next

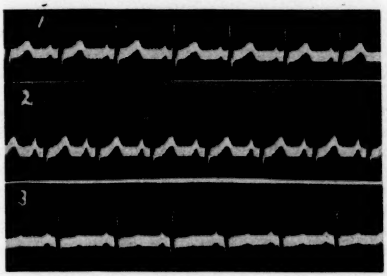

FIGURE 2. *Electrocardiogram after the Administration of Vertavis Showing Normal Sinus Rhythm in All Three Limb Leads.*

tablet of vertavis on July 24 produced normal sinus rhythm for only 3½ hours. Another tablet of vertavis that afternoon caused only a transient conversion to sinus rhythm, with frequent changes from sinus rhythm to paroxysmal auricular tachycardia throughout the night. On July 25, 26, 27, 30 and 31 vertavis was given — at first one tablet in the morning and one in the afternoon, and then one tablet followed within 1 hour by another; there was no conversion to sinus rhythm.

On August 4 and 5 0.003 and 0.006 gm. of pilocarpine were given without effect. On August 6 physostigmine (0.0006 gm.) produced no effect; later 0.0012 gm. was given without effect. On the next day a blood-pressure cuff was inflated about the patient's neck for about 20 seconds without response. On August 9 magnesium sulfate — 20 cc. in a 10 per cent solution — was given intravenously without producing a change. On August 11 one tablet of vertavis was given at 9 a.m. and a second at 1:15 p.m., without effect.

Mecholyl in doses from 5 to 7.5 mg. was given on August 18 and again on August 22, with a change in the rhythm to sinus tachycardia, but no change to normal sinus rhythm. Each time the sinus tachycardia was soon replaced by the paroxysmal auricular tachycardia.

A tablet of vertavis was tried again on October 5, without effect.

All the routine laboratory studies, including blood studies (red-cell count, white-cell count, sedimentation rate and cholesterol), urinalyses and basal metabolic rate, as well as x-ray films of the chest, were normal.

The patient is now attending school without difficulty. The pulse continues to be rapid and regular.

DISCUSSION

The etiology of similar cases has been quite varied. Diphtheritic myocarditis usually occurs from the sixth to the twenty-first day after the onset of diphtheria.[17] It was the opinion that the case reported above probably did not represent a diph-

theritic myocarditis because of the late time relation and the lack of other signs or symptoms of myocarditis. In a series of 100 cases of severe diphtheria Begg[18] took routine electrocardiograms and found 1 case of paroxysmal auricular tachycardia. That case was fatal before the nineteenth day of the illness. Anderson[19] reported the case of a thirty-nine-year-old man who developed paroxysmal auricular tachycardia six weeks after a severe case of diphtheria and died within seventy-two hours of the onset of the tachycardia. In this case, however, it is probable that the patient had myocarditis much earlier.

Similar cases have been uniformly resistant to therapy. No reports in the literature state that veratrum viride has been used successfully in such a case. In a clinicopathological conference reported from the Massachusetts General Hospital[20] the unsuccessful use of the tincture of veratrum viride is mentioned. Collins[21] also reported the unsuccessful trial of veratrum viride tincture in a case of paroxysmal auricular tachycardia. The drug is not without danger and should only be used with a full knowledge of its toxic effects. The progressively diminishing response to vertavis, which probably represents tachyphylaxis, has been observed in dogs by Krayer and Acheson.[22]

Veratrum viride is obtained from the rhizome and roots of the plant *Veratrum viride*, commonly known as the green hellebore. It contains a mixture of alkaloids, the most important of which is protoveratrine. The main actions of veratrum are slowing of the heart rate, a fall in systemic blood pressure, slowing of the respiratory rate and a "collapse reaction" characterized by sweating and reduction in body temperature. Poisoning by the drug is characterized by nausea and vomiting, diarrhea, headache and cardiovascular collapse. Large doses of veratrum are rapidly vomited, however, so that death is rare. The site of action of the drug is believed to be the vagal nuclei in the medulla and the afferent nerve endings of the vagus nerve. Atropine will abolish the effects of veratrum and should be at hand in the event of an overdose.

Summary

A case of prolonged paroxysmal auricular tachycardia in a five-year-old boy two months after a moderate attack of diphtheria and two weeks after an attack of varicella is presented. The paroxysmal tachycardia has persisted to date except for occasional, transient spontaneous conversion to normal sinus rhythm and has been temporarily converted to sinus rhythm by mecholyl and veratrum viride.

Digitalis, quinidine, potassium acetate, potassium chloride, physostigmine, pilocarpine, magnesium sulfate, carotid-sinus pressure, eyeball pressure, sedatives and a blood-pressure cuff inflated about the neck were without effect on the tachycardia. The patient has been asymptomatic throughout the duration of the tachycardia and is leading an active life. At no time has he shown any signs of cardiac enlargement or decompensation.

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FOOD SENSITIVITY IN 100 ASTHMATIC CHILDREN*

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THAT food can cause asthma in children is a fact beyond dispute. The frequency of this sensitivity, as well as its relative importance to recurrent respiratory infection, sensitization to pollens and other environmental allergens, is an entirely different question, concerning which there is considerable difference of opinion.

An example of the way allergy is not infrequently treated is provided by the history of an asthmatic child of five years who was recently seen. She had previously been treated in another city and brought with her a typed list, bound in the form of a small book, of the foods that she could and could not eat, as determined by skin tests. The "good" foods were typed in black, and the "bad" foods in red; it was all very impressive. She had been tested with two hundred and thirty-six foods, including squab, tripe, calves' brain, venison, caviar, poppy seed, elderberry, pheasant, hops, chives and Limburger cheese, and had been found allergic to one hundred and nine of them, including Sanka coffee, rutabaga, German celery, thyme, cranberry, sand dab, tripe, sweetbreads and watercress. Among the foods she was allowed to eat were pheasant, guinea hen, venison, horse meat, turtle, abalone, terrapin, caraway seed, juniper, avocado, mango and quince. The asthma was in reality due to grass and tree pollen, and she was later quite successfully hyposensitized, with no dietary restrictions whatever.

This is by no means an isolated example. Too many people, without proper training or common sense, treat allergy in this way. Food allergy in particular lends itself to exploitation, for a dietary "cure" of any sort is a form of therapy that has made a great impression on the laity from time immemorial, and some of the greatest fads and misconceptions in medicine have had "diets" as their basis.

That sensitization to food is a common cause of allergic symptoms is certainly true; that too much pseudo-science and too little common sense are too often used in dealing with foods as allergens is equally true:

> The general interest in diets as such; the tendency to attribute to the ingestion of foods all manner of complaints; the deep impression made by the occasional instance in the asthmatic of the sudden, sharp, often dangerously severe attack following the eating of a specific food, combine to give undue consequence to food allergy as a cause of asthma.[1]

To determine the influence of food upon asthma is by no means easy because there are so many extraneous complicating factors involved, and no statistics can be really accurate.

For the purposes of this study, 100 asthmatic children between the ages of three and twelve years, partly from the clinic and partly from private practice, were observed and were tested by the scratch method. Thirty-five gave one or more positive tests to foods, and 65 gave none. There was no reason to believe that asthma was caused by food in any of those who gave negative tests; the asthma could be accounted for in other ways (such as animal emanations, pollens and upper respiratory infections). It is recognized, however, that asthma may be rarely caused by a food and that there may still be a negative skin test: the bronchial mucous membrane is sensitized, and the skin is not. In children this situation is believed to be rare; it is hardly ever necessary to employ elimination diets in the presence of negative skin tests to determine food sensitivity in the study of an asthmatic child. It is also recognized that an intracutaneous test to a food is often positive when the scratch test is negative. It was formerly the custom in this clinic to do routine intracutaneous tests with foods in all the asthmatic children. There were so many positive reactions that meant nothing clinically and it was so rare to obtain any information of value that this procedure was given up. Intracutaneous tests with environmental allergens, particularly with pollens, when scratch tests are negative, are, however, of great value, and when positive usually indicate clinical sensitivity.

Very few of the children in this series had continuous asthma — in almost all there were periods of at least three or four weeks when they were free from it. If they were eating the foods in question daily or frequently during these periods and no asthma was produced, it was concluded that the foods were harmless so far as asthma was concerned. If the child was having nearly continuous asthma, the reacting foods were removed from the diet and reintroduced one at a time during asthma-free periods, and the results noted. If there was any suspicion of a food that was eaten infrequently, this was given in ordinary amounts, and if no asthma occurred within forty-eight hours, it was concluded that the food in question had nothing to do with the asthma. There were a few cases of very large skin reactions to fish or nuts in which this test was not done, since I did not wish to run the risk of producing a dangerous reaction with these potent allergens. The food question was discussed frequently and for considerable periods with the mothers, for most of the children were being brought

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in for inoculations each week. It was almost always found that the mother knew from previous experience what foods produced asthma.

In 24 of the 100 children studied asthmatic attacks had been or could be produced by the ingestion of a specific food. In most cases this had been recognized by the mother before the child was seen, and the food or foods in question had been removed from the diet, so that there were not many cases in which food was concerned in producing the asthma at the time the child was brought in.

In the 100 children there were 218 positive scratch tests to foods (Table 1). Of these, there were 44 (20 per cent) in which it was definitely proved that the food in question could cause asthma. It is especially noteworthy that egg white, fish, peanut, walnut and chocolate accounted for 38 of the 44 etiologic tests. The other foods producing asthma were tomato in 2 cases, spinach in 1, orange in 1, corn in 1 and barley in 1. Although potato gave 22 positive tests (some of them large wheals with pseudopods), in no case could it be shown that the ingestion of potato produced asthma or any other symptom. Since the first days of skin testing, spinach extracts have always been nonspecifically irritating, and it is probable that a large proportion of the positive tests to spinach were of this character. It did cause asthma in 1 patient, however. In 16 cases (8 per cent) a food that gave a positive test caused irritation about the mouth, vomiting, urticaria or angioneurotic edema, but no asthma. This was true of asparagus and banana in 1 case each, egg white in 4, fish in 2, orange and peanut in 1 each, spinach in 2 and walnut in 2. No symptoms were caused by one hundred and fifty-eight foods (72 per cent) to which tests showed skin sensitivity. It is probably true that asthma would have been produced in some of these patients had a considerable amount of the food been ingested. Not uncommonly, however, the degree of sensitivity to some of these foods, such as fish and nuts, is so high that as soon as any of the food touches the lips or enters the mouth, the mucous membrane begins to swell, and the food is immediately spat out or regurgitated before there has been any chance for it to be absorbed. If these cases are included, the number of foods producing asthma is increased from forty-four to sixty.

TABLE 1. *Results of Skin Tests to Foods in 100 Asthmatic Children.*

FOOD	TOTAL NO. OF POSITIVE TESTS	ASTHMA	VOMITING AND HIVES*	NO SYMPTOMS
		NO. OF CASES	NO. OF CASES	NO. OF CASES
Apple	3			3
Asparagus	4		1	3
Banana	5		1	4
Barley	5	1		4
Beef	4			4
Carrot	0			
Celery	6			6
Chicken	8			8
Chocolate	5	4		1
Corn	5	1		4
Egg white	21	6	5	10
Fish	25	12	2	11
Lamb	3			3
Milk	1			1
Oat	4			4
Orange	5	1	2	2
Peanut	12	10	1	1
Pea	6			6
Pork	2			2
Potato	22			22
Rice	3			3
Rye	8			8
Spinach	27	1	1	25
String bean	6			6
Tomato	9	2		7
Wheat	5			5
Walnut	13	6	3	4

*No asthma caused by foods in these cases.

DISCUSSION

These figures agree with those of Adams[2] and of Chobot and Hurwitz.[3] Adams found that of 130 positive scratch tests to foods in a group of allergic children, 23 per cent were of clinical significance. Chobot and Hurwitz observed that 18 per cent of 198 positive scratch tests to foods in 38 allergic children were of clinical significance.

It seems well established, therefore, that only about a fifth of the positive scratch tests to foods in asthmatic children have anything to do with the asthma, and furthermore that although it is possible for clinical sensitivity to any food to exist in these children and to be at least a contributing factor to the asthma, the number of foods that cause such sensitivity with any frequency is not large (fish, egg, nut, peanut and chocolate); as a rule, a high degree of sensitivity must exist for a food to cause asthmatic attacks. Wheat and milk, which may be of considerable importance in the atopic dermatitis of infants and in many other allergic conditions, rarely caused asthma in the children under consideration, and positive scratch tests were not common (Table 1).

In asthma, positive skin tests to environmental allergens are usually of etiologic significance: the mucous membrane of the respiratory tract, as well as the skin, becomes easily sensitized to these allergens. To cause a positive skin test to a food, unsplit food protein must at some time have entered the blood stream, and in atopic people an immunologic reaction results, which is manifested by a positive skin test. The respiratory mucous membrane is likely to escape sensitization; the immunologic reaction is shown only on the skin, and possibly by the presence of reagins in the blood serum. Positive tests to foods have the same significance as positive tuberculin, trichophytin or brucellergen tests: at some time the organism has been exposed to antigenic material, and an immunologic reaction, which may or may not have anything to do with the clinical condition under consideration, has occurred. One would expect, however, that if there were a high degree of skin sensitivity, as in many of the nonetiologic tests, if the food were eaten,

dermatitis or urticaria would result, inasmuch as the skin is sensitized. Nevertheless, in 218 cases of positive tests in the series of children under discussion, the corresponding food could be eaten with complete impunity in 158 — there was no asthma, urticaria or eczema, in spite of the fact that in many of these patients the skin showed a high degree of sensitization.

Some of the positive tests had been of etiologic significance when the child was younger, and with increasing age he had become "tolerant" to the food; some of them had never been of etiologic significance. There is a great tendency in allergic children to acquire tolerance to foods as they grow older; many children who have violent symptoms from egg white at the age of a year, can eat it with impunity at the age of six years, but the positive skin test often persists, and the reaction may be as large as it was in the beginning. Tolerance to environmental allergens does not come so readily: the child clinically sensitive to cat hair or to ragweed pollen is likely to remain sensitive for a long time, often through life.

The immunologic situation in these children who are tolerant to foods in the presence of large positive skin reactions, and who often have reagins of high titer as well, is not clearly understood, nor is the mechanism by which they acquire this tolerance. If absorbed food protein reaches the skin, urticaria or dermatitis should result. Therefore, either unsplit food protein is not absorbed in sufficient quantity to cause a reaction, or some mechanism has been developed by which it is rendered innocuous after it has been absorbed. It must be remembered, however, that a very high degree of skin sensitivity must be present before urticaria can result from an ingested food, and that the solutions used by outside application or intracutaneous introduction in testing, which produce whealing reactions, are probably many times more concentrated than the concentration of a small amount of absorbed food protein when it has been diluted in the several liters of blood contained in the body. If the degree of skin sensitivity is very high, however, an infinitesimal amount of absorbed allergen may cause whealing. Brunner and Baron[4] found, for example, that the intravenous administration of cottonseed extract containing 0.0001 mg. of nitrogen caused an urticarial reaction on skin sites that had been artificially sensitized with a serum containing a high titer of cottonseed reagin. When diluted with the amount of blood plasma contained in an adult (approximately 3000 cc.), this would mean a dilution of 1:30,000,000. Walzer[5] found that 0.0001 mg. of nitrogen represented 1/5600 of one cottonseed. So that with a very high degree of skin sensitization it is necessary for only traces of food protein to be absorbed to cause urticaria.

It is a fact that any food protein can be absorbed unsplit — if this were not so there would be no positive skin tests to foods. It is also a fact that this absorption can happen at any age and probably true that there is a considerable difference in the ease with which various food proteins are absorbed — for example, fish, nut and egg protein are consistently absorbed by many people. Not much is known about other foods, however, and it is not unlikely that with many people the unsplit protein of many foods is absorbed only for a short time when the food is first eaten, enough for the development of an immunologic reaction, evidenced by a positive skin test — no more after that, or at any rate not enough to cause a reaction except possibly if an excessive amount of the food is eaten. Some people, however, seem to absorb the unsplit protein of a number of foods over long periods: if this were not so there would be no asthma from food, no dermatitis, no urticaria and no migraine. It seems likely that there is a great individual variability in this respect.

If unsplit food protein reaches the skin in sufficient amount and the skin is strongly sensitized, some skin manifestation should result. Therefore if no reaction takes place, the protein has not been absorbed in sufficient amount, or at all, or it has been changed after absorption in some way so that it is no longer antigenic.

In asthma the situation is somewhat different, for the bronchial mucous membrane may not be sensitized. If it is sensitized, the situation in asthma may be compared to that in dermatitis: either failure of sufficient absorption or neutralization after absorption.

Conclusions

About a fifth of positive scratch tests to foods in asthmatic children are of etiologic significance.

The mother usually knows what food produces asthma, and has omitted it before skin tests are done.

Theoretically, any food can cause asthma; practically, not many foods commonly do so. Fish, egg, walnuts, peanuts and chocolate are the most important. Wheat and milk can cause asthma in children, but do not often do so.

Sensitivity to food, which should always be taken into consideration in asthmatic children, is of relatively little importance in comparison with sensitivity to pollen, other environmental allergens and upper respiratory infections.

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GENERALIZED EXFOLIATIVE DERMATITIS DUE TO PENICILLIN*

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THE increasing frequency of reactions to penicillin warrants wider recognition of the dangers of the local application of penicillin, and more reliance placed on less sensitizing methods of administration, such as the intramuscular, intravenous and intra-arterial routes.[1, 2] Although the direct toxic action of penicillin is negligible,[3] aside from the irritating effect when the drug is applied to the central nervous system,[4] the delayed or acquired sensitivity reactions may be severe or fatal. Wile[5] noted that severe allergic reactions involving edema of the pharynx, larynx and lungs complicating penicillin therapy are serious and may result in fatal complications. Numerous other authors have reported severe reactions simulating asthma,[6] serum sickness[7, 8] and epidermal reactions such as bullous dermatitis.[9] Kolodny and Denhoff[10] have stated that patients with dermatologic disorders have a higher sensitivity than other patients. Gottschalk and Weiss[11] noted that a delayed or acquired sensitivity could be produced by repeated local applications of penicillin. By using a simple patch test they were able to sensitize 4.5 per cent of 200 persons to penicillin ointment. Pillsbury[12] states that the incidence of sensitivity reactions at present is over 15 per cent, and he believes that this percentage may increase as the incidence of exposure to penicillin is increased. The fact that these reactions are not limited to local manifestations but that severe generalized eruptions occur often escapes due consideration. Cormia[13] noted severe systemic involvement in patients after the local application of penicillin, and Barksdale[14] reported a fatal case of exfoliative dermatitis due to penicillin therapy. Barksdale's patient did not have syphilis and had become sensitized to penicillin therapy while at sea. He was also sensitive to sulfonamides. He was given 100 units of penicillin every three hours, for a cellulitis of the skin, but at the end of the seventh dose the administration of penicillin had to be discontinued because of a severe cutaneous eruption, which progressed into a generalized exfoliative dermatitis, streptococcal septicemia and death. Because of this and other toxic reactions complicating the use of penicillin locally for the treatment of skin diseases, Barksdale concludes that "penicillin locally in any form is absolutely contraindicated." The following case is reported as further evidence that complications dangerous to life may result from the local use of penicillin.

CASE REPORT

A 27-year-old man with no history of allergic disorders or previous penicillin therapy, was seen in the outpatient clinic of an Army hospital ship on November 14, 1946.

Physical examination disclosed typical impetigo contagiosa of the skin, with honey-like crusted lesions on an erythematous base on the left side of the neck.

The patient was instructed to wipe the area twice daily with alcohol and after this had dried to apply penicillin ointment in a petrolatum base (1000 units per cubic centimeter) locally. The lesions retrogressed and the patient improved during the first 3 days of therapy, but on the 4th day coalescing vesicular lesions covered with a honey-like material on an erythematous base spread further on the left cervical region and appeared at the right angle of the mouth. He was admitted to the hospital, and local therapy was continued with penicillin ointment. Two days later the individual lesions had improved, but an erysipelatous lesion had appeared involving the skin more deeply and covering the entire area over which the ointment had been applied. This sharply demarcated area was erythematous, edematous and indurated, and there was an increase in local temperature. There was no systemic toxicity, however, to suggest a true erysipelas. Local penicillin therapy was discontinued, and Burrows boric acid wet soaks were given as the only treatment. On the following day the erysipelatous lesions had spread further on the neck so that 30,000 units of penicillin every 3 hours was started intramuscularly in addition to the local wet compresses. No improvement of the local lesions resulted, and on November 22 severe pruritus of the involved area occurred. On November 23 a fine maculopapular generalized eruption was noted with a more erythematous reaction over the scrotum and inner aspects of the thighs. A diagnosis of dermatitis medicamentosa was made, and the intramuscular administration of penicillin was discontinued immediately. Phenolated zinc lotion was used to control the intense associated pruritus. The skin lesions deepened in color, and within the next 7 days a generalized exfoliative reaction of the entire body, including the palms, soles, scalp and eyelids, occurred.

The patient subsequently had no difficulty, but he was cautioned against receiving penicillin in any form again. An interesting therapeutic note was that the impetigo of the face and neck never disappeared on either local or intramuscular administration of penicillin, but disappeared after 5 days of therapy with ammoniated mercury ointment.

DISCUSSION

This case demonstrates that systemic sensitization to local applications of penicillin may rapidly occur so that severe generalized reactions are not prevented by a change to parenterally administered penicillin. The potential dangers of penicillin applied locally more than offset any advantages of this mode of therapy. Generalized exfoliative dermatitis is a complication to be feared and should not be risked by the local application of penicillin ointment to an innocuous skin lesion. In the case reported above continuation of intramuscular penicillin after the occurrence of the generalized rash might have caused a fatality. The occurrence of localized exfoliative dermatitis in other cases has emphatically illustrated the dangers of local penicillin. One patient had an exfoliation of the skin of the entire external ear canal and pinna, after treatment of a chronic suppurative otitis media, with purulent drainage from a tympanic perforation by installation of penicillin solution into the ear. The impression gained from personal experience is that the local administration of penicillin in the region of the head and neck causes a higher rate of sensitization and is definitely contraindicated. With

*The opinions and views set forth in this article are those of the writer and are not to be considered as reflecting the policies of the Army Medical Department.

the possibility of severe and potentially fatal reactions and the chance that sensitivity induced by local therapy may prevent parenteral penicillin therapy when a severe systemic infection later occurs, it seems best to discontinue local penicillin therapy entirely. The availability of other suitable local therapeutic agents, such as ammoniated mercury and tyrothricin, and the superiority or equality of parenterally administered penicillin in the treatment of superficial cutaneous infections are further facts to discourage the dangerous practice of using penicillin locally.

Summary

A case of generalized exfoliative dermatitis due to sensitivity to penicillin is presented.

Penicillin ointment is contraindicated in the treatment of skin lesions because of the reactions dangerous to life that may occur during the treatment of an innocuous condition and also because systemic sensitization may prevent later parenteral use of penicillin when it is needed to combat a severe systemic infection.

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MEDICAL PROGRESS

REGIONAL ANESTHESIA*

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THE war and the following years witnessed a widespread increase of interest in regional anesthesia. This was a natural sequence to the emphasis placed upon the role of anesthesia in war surgery. Many of the young physicians who received training in anesthesiology during the war have chosen to complete their training in that specialty. Their enthusiasm has added to the magnitude of experimental and clinical investigation of the postwar period. Differential spinal block has furnished new evidence of the mechanism of nerve block. New diagnostic and therapeutic sympathetic blocks have broadened the field of the anesthesiologist. Segmental spinal anesthesia invites further investigation into the potentialities of decreasing the concentration of a spinal anesthetic agent. The synthesis of morphine-like compounds promises a brighter future for better premedication and postoperative relief of pain. The development of new and the modification of older technics in the past few years have aided in the personalization of the anesthesia to the individual patient.

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Differential Spinal Block

It has long been known that anesthetic agents introduced into the subarachnoid space did not equally affect all nerve components. New evidence that the smaller, nonmyelinated fibers are blocked first is presented in the work of Sarnoff and Arrowood[1-3] on "differential spinal block." They employed the continuous spinal technic with 0.2 per cent procaine hydrochloride solution in an attempt to produce differential block of the sympathetic fibers. Skin-temperature readings were used as a measure of sympathetic activity. Their results indicated that sympathetic preganglionic vasomotor and sudomotor fibers were blocked at or about the same time as the fibers concerned with the appreciation of pinprick. These fibers were blocked without impairment of the modalities of touch, deep pressure, position sense or vibratory sense and with no loss of motor power. A further observation was that position sense and the stretch reflexes are mediated by separate groups of fibers. This was shown by a loss of abdominal, patellar and Achilles-tendon reflexes when position sense was still appreciable.

Sarnoff and Arrowood noted a drop in blood pressure with sympathetic spinal block equal to that observed after classic spinal block. From this they concluded that muscular flaccidity and diminished thoracic excursions play little part in the hypotension seen with spinal anesthesia.

This hypotension resulting from sympathetic vasomotor block is to be differentiated from the drop in blood pressure seen on intra-abdominal manipulation as described by Rovenstine et al.[4] This group demonstrated a fall in blood pressure coincident with rapid change in position of the patient, abdominal incision, handling of viscera and the use of abdominal packs.

Further light on the action of local anesthetic drugs is found in the work of Bennett and Chinburg.[5] Studying the resting and demarcation potentials in isolated sciatic nerves under a variety of local anesthetic drugs in frogs, they found that conduction could be blocked without depolarization of the nerve. They concluded that the mode of action of these drugs was an infinite prolongation of the refractory period and that depolarization was not needed for complete block.

Sympathetic-Nerve Block

Sarnoff and Arrowood[2] have used differential spinal block as a preoperative prognostic test in patients with hypertension or peripheral vascular disease. Russek et al.[6] employed high caudal block for the selection of hypertensive patients for sympathectomy. Evans[7] and Luke[8] reported on paravertebral sympathetic block as a preoperative test for patients with peripheral vascular disease.

The variety of indications for the use of sympathetic-nerve blocks was aptly reviewed by Faust,[9] who discussed both diagnostic and therapeutic uses. Sympathetic block has been extensively employed for the relief of pain of the causalgia syndrome. Freeman[10] and Rasmussen and Freedman[11] reported on two series of post-traumatic causalgia in Chinese soldiers. They found that procaine paravertebral sympathetic block, single or repeated, gave temporary to permanent relief. Ulmer and Mayfield[12] described dramatic temporary relief of causalgia from sympathetic block. They stated, however, that the principal value of block in this disorder is to permit more extensive examination and evaluation of the injury, and were not impressed with any permanence of relief. Shumacker et al.[13] reported permanent relief of the pain of causalgia in 25 per cent of 83 patients with procaine sympathetic blocks alone. They stated that patients whose favorable response to the first injection was limited to the length of sympathetic anesthesia had a poor prognosis for permanent relief by block alone. Patients who had a prolonged period of relief from the initial injection were the ones most likely to obtain permanent relief from sympathetic blocks. Tyson and Gaynor[14] found paravertebral sympathetic block of value in post-traumatic painful and spastic states and vascular injuries and for the relief of pain associated with acute fractures and crushing injuries of the extremities. Fitzpatrick and Adams[15] stated that paravertebral and intercostal nerve block allows for more adequate coughing and ventilation in patients with thoracic injuries. Rasmussen and Alessi[16] reported two sympathetic blocks lasting several weeks when rectocaine (5 per cent benyl-alcohol, propylaminobenzoate and procaine) was injected by the paravertebral route.

The treatment of acute thrombophlebitis by paravertebral sympathetic nerve block as popularized in this country by Ochsner[17] is a well established form of therapy. It not only interrupts the vasospasm and relieves the pain attendant upon this condition but also allows for movement of the limbs to aid the collateral circulation in becoming effective. Benson[18] reported on the use of continuous caudal anesthesia to provide prolonged and uninterrupted sympathetic block. He claimed good results in the management of acute post-partum thrombophlebitis of the lower extremity. Ellis and Sheffrey[19] used the same technic in acute pelvic thrombophlebitis.

That the value of sympathetic-nerve block as a method of therapy is increasing is evidenced by the number of disorders in which it has proved effective. Risteen and Volpitto[20] performed stellate-ganglion blocks so as to provide maximal vasodilatation of the intracranial arteries in patients with intracranial hemorrhage, embolism, thrombosis and arterial spasm. In three thousand blocks on 275 patients they reported good results in arterial spasm and sufficient improvement in the other disorders to warrant the continued use of stellate-ganglion block as a therapeutic measure.

Bageant and Raper[21] reported immediate relief of symptoms and improvement after stellate-ganglion block in 2 patients with pulmonary emboli. Lindroth[22] has interrupted the splanchnic nerves by paravertebral injection of absolute alcohol with the patient anesthetized by cyclopropane to control the pain of the injection. There are two reports in the Russian literature on cervical-vagus-sympathetic block. Vishnevskiy[23] was able to prevent the development of reflex manifestations in the respiratory and cardiovascular systems during the course of pneumonectomies in dogs. Skvostsov[24] produced relief of pain and freedom from dyspnea in war-casualty patients with open pneumothorax. He also found that cervical-vagus-sympathetic block was followed by a rise in blood pressure in patients in whom shock had developed. Braun,[25] in a study on a small group of patients with angina pectoris, provided relief of pain and electrocardiographic improvement with paravertebral thoracic sympathetic block. He ascribed the relief of anginal pain to interruption of the sensory pathways. The

improvement in the electrocardiogram was explained as evidence of increased coronary blood flow.

Collins et al.[26] used paravertebral sympathetic block in the management of vasomotor disturbances in poliomyelitis. Their work was done on patients who in the convalescent period of the disease exhibited cyanosis or discoloration of the skin accompanied by mild to severe edema and hyperhidrosis. Their results varied from partial to complete relief.

Trimble and Sanford[27] reported on the relief of sulfadiazine anuria by procaine block of the celiac ganglion in 1 patient. They also mentioned relief of ureteral colic from ureterolithiasis with the same block. Newell[28] described the case of a patient who had anuria due to sulfadiazine for seventy-two hours and who was being prepared for nephrostomy in which a spinal anesthetic agent was given, and just before the drapes were placed urine was seen to flow from the indwelling urethral catheter. The operation was canceled, and the patient made an uneventful recovery.

Segmental Anesthesia

There has recently been a trend among anesthesiologists to decrease to a minimum the amount and concentration of the spinal anesthetic agent within the subarachnoid space. By this it is hoped that the postanesthetic recovery period will be shortened, with a correlated decrease in postoperative complications, and that the incidence of neurologic complications will likewise fall with a lowered concentration of the spinal drug. The ease of obtaining general anesthesia with pentothal and the acceptance of curare as a muscle-relaxing agent have made possible the clinical investigation of dilute solutions and new technics without jeopardizing adequate anesthesia. Solkow[29] reported on the use of dosages of 3 to 5 cc. of 0.25 per cent metycaine with hypertonic glucose to produce selective sensory block. Fisher and Whitacre[30] employed 2-mg. to 6-mg. doses of 1:2000 to 1:6000 dilutions of nupercaine.

Saklad et al.[31] have introduced a new technic for segmental anesthesia utilizing a catheter placed in the subarachnoid space. They passed a Tuohy continuous spinal catheter through a Huber pointed needle for a predetermined distance cephalad in the subarachnoid space. Injection of the anesthetic agent through the catheter then allowed for placement of the anesthetic at the level of the spinal segments desired. Dilutions of pontocaine with spinal fluid in amount of 1:1000 and 1:2000 were found sufficient for adequate anesthesia by this technic. Saklad stated that upper abdominal anesthesia may be complete without loss of motor power of the lower extremities. Movement of the legs or merely maintenance of normal muscle tone in the legs aided in the maintenance of preanesthetic blood-pressure levels. Saklad postulated that the retention of motor power in the lower extremities might be expected to lower the incidence of postoperative thrombophlebitis.

New Drugs

Vasopressors

More and more attention is being directed toward drugs that can be used to maintain blood pressure during spinal anesthesia. D-desoxyephedrine (methedrine), a relatively new member of the aromatic amine family, has been given extensive trial with promising results. In a comprehensive report on methedrine, Anderson[32] called attention to its prolonged sustaining effect in its optimal 20-mg. dose. Its pressor effect is peripheral, but larger doses have a strengthening action on the cardiac musculature. The toxicity of methedrine is one eighth to one tenth that of ephedrine, weight for weight. In larger doses it is claimed to have a stimulating effect upon respiration.

Melville[33] studied the pressor activity of varying concentrations of ephedrine and pitressin administered in repeated doses. He concluded that the most effective combination was a mixture containing a ratio of 4.8 mg. of ephedrine to 1 unit of pitressin. Chaikoff,[34] in an extensive review of the clinical use of vasopressor drugs with spinal anesthesia, concluded that the combination of ephedrine and pitressin was superior to ephedrine alone as a prespinal prophylactic drug and that this combination should be administered eight to ten minutes before the spinal puncture. Dripps and Deming[35] investigated the pressor-maintenance value of several sympathomimetic amines in clinical prespinal doses. They found that methedrine and the pitressin-ephedrine combination were superior to paredrine or ephedrine alone in preventing the hypotension associated with spinal anesthesia.

A new series of pressor drugs is now under investigation, but as yet sufficient evidence is not available to warrant their general acceptance. These are the aliphatic or straight chained amines whose vasopressor responses suggest that they produce sympathetic stimulation. Roman-Vega and Adriani[36] selected oenethyl (2,-methyl-amino-heptane) for clinical trial with spinal anesthesia. After using it in 1000 surgical patients in dosages of 75 to 100 mg. given intramuscularly, they stated that it is worthy of further trial. Le Compte,[37] reporting a small series, was also favorably impressed. Ahlquist,[38] experimenting with dogs, described a tachyphylactic reaction with repeated doses of the aliphatic amines evidenced by a depressor response preceding the pressor action. Squires[39] described 2 cases of skin slough following the clinical use of oenethyl mixed with pontocaine given subcutaneously in the lumbar area. He was able to produce sloughing repeatedly at the site of injection when oenethyl was mixed with pontocaine and injected subcutaneously in guinea pigs.

Jackson[40] described another aliphatic amine similar to oenethyl except for an extra methyl and hydroxyl radical. This drug, termed EA–83, is readily absorbed from the gastrointestinal tract in dogs, but as yet no clinical investigation has been undertaken.

Morphine and Morphine-like Drugs

Drew, Dripps and Comroe[41] studied the effects of morphine upon the circulatory changes in man after position change. They found that movement of a "morphinized" patient from the supine to the sitting or semierect position may be followed by vascular collapse. Powers[42] re-emphasized the addiction properties of demerol, and Robbins[43] demonstrated that cyclopropane-induced cardiac irregularities after morphine premedication are not seen in the dog when demerol is used. Foster and Carman[44] tested demerol and other piperidine derivatives on the rat and reported that some are deserving of clinical trial.

When the United States Government study commissions went to Germany after World War II, they found a group of compounds, prepared by German chemists, that exhibited hypnotic action. One of these synthetic hypnotics — No. 10820, or dolophine — has been thoroughly studied by the Lilly research group and named methadon (6-dimethylamino-4-4-diphenyl-heptanone-3). C. C. Scott and Chen[45–47] found that methadon has an analgesic effect as powerful as morphine. In addition, it depressed respiration, caused cardiac slowing and produced a slight to moderate fall in blood pressure, but there was no depression of salivary secretion and it only occasionally caused nausea and vomiting. An associated depression of the cough reflex and parasympathomimetic stimulation of the small bowel were noted. Isbell[48] used methadon in the treatment of morphine addiction and reported the development of tolerance and addiction to methadon. With methadon, however, withdrawal symptoms were not nearly so severe as those with morphine, and he advocated substitution of methadon for morphine before withdrawal in addicts. W. W. Scott et al.[49] observed no euphoria when methadon was given clinically for preanesthetic medication and postoperative relief of pain.

Local Anesthetic Agents

An increasing number of compounds are being investigated from the standpoint of their potential anesthetic properties. Krop[50] studied pyridium (β-phenylazo α-α' diamino pyridine monohydrochloride) and re-emphasized that it might be useful as a topical anesthetic in the bladder and urethra. Kuna and Seeler[51] studied the anesthetic properties of a large group of alkyl amino alcohol esters and reported that several hold promise for infiltration anesthesia. One in particular in this group, 2-cyclopentyl amino-1-propyl p-aminobenzoate hydrochloride, might prove valuable for topical as well as for infiltration anesthesia. Ramsey and Haag[52] reported a series of aryl-urethanes that possessed anesthetic activity greater than that of cocaine when instilled in the rabbit eye.

Combes et al.[53] advocated the use of topical anesthetic agents in ointment bases for the treatment of burns and cutaneous ulcers. They claimed that this procedure promoted rapid healing and soft scar formation by lessening the vasodilatation, edema and muscle spasm that accompanies the pain of the acute lesion.

Obstetric Anesthesia

Spinal analgesia for obstetric delivery is enjoying greater popularity than ever before. Weaver and his co-workers,[54] Turner[55] and Marcus and his associates[56] reported on large series with excellent results. Cullen and Griffith[57] compared post-partum results of spinal and gas anesthesia for vaginal delivery. The evidence in their study was in favor of spinal anesthesia, except that headaches were severer, although the incidence of headache in the two groups was the same. Weintraub et al.[58] compared the incidence of postlaparotomy spinal headache in cesarean section and various gynecologic operations with the incidence in vaginal delivery under spinal anesthesia. The incidence of headache following spinal anesthesia was much higher in the last group than in the first two groups, averaging 15 per cent in 300 patients. In the vaginal-delivery group 50 per cent of the patients who complained of headache exhibited orthostatic hypotension or tachycardia or both. These investigators found that the application of a tight abdominal binder post partum gave complete or almost complete relief in 90 per cent of the patients with headache.

Resnick,[59] of Great Britain, weighted nupercaine with glucose for use in obstetric delivery. Parmley and Adriani,[60] in this country, popularized "saddle block" anesthesia with heavy nupercaine for vaginal delivery.

Caudal and continuous caudal anesthesia are still enjoying great popularity in certain communities. Brown et al.[61] tested various concentrations of pontocaine in physiologic saline solution with epinephrine (dilution of 1:200,000) for duration of effect. Using continuous caudal anesthesia, they found that pontocaine, 0.15 per cent, proved the most effective.

A review of the technic of pudendal-nerve block is reported by Eisaman and McHenry.[62]

Technics

The increasing number of qualified anesthesiologists has brought reports of new technics in regional anesthesia. Adriani and Roman-Vega[63] used saddle-block anesthesia with a weighted solution of nupercaine. They employed the sitting position for injection and produced anesthesia in the anoperineal region without motor block of the lower extremities.

A method of continuous brachial-plexus block utilizing a blunt needle with a cork stabilizer was presented by Ansbro.[64] Kershner and Shapiro[65] reported a technic of interlaminar approach for spinal subarachnoid puncture as an alternative to the conventional interspinous method.

Lund and Rumball[66] dissolved pontocaine crystals in distilled water to make a 0.1 per cent hypobaric solution for which they claimed up to two and a half hours of analgesia. The addition of epinephrine gave a prolongation of 30 to 50 per cent. Potter and Whitacre[67] added ephedrine to a pontocaine-glucose solution in a controlled series and concluded that the ephedrine decreased the dosage by approximately a third but did not appreciably increase the length of its effect. Ruben[68] added ephedrine to pontocaine glucose and made use of the prolonged lateral position to obtain increased length of anesthesia for unilateral orthopedic procedures on the lower extremity.

Roman-Vega and Adriani[69] reported further on the use of weighted nupercaine with gravity control for general abdominal surgery. Nitikman[70] outlined another formula for nupercaine dosage in nucleus-pulposus surgery.

Two articles on the status of epidural anesthesia are in the recent literature, both from the southern continents where this technic apparently enjoys much greater popularity than it does in this country. Lins[71] reported from Brazil, and van Hoogstraten[72] from the Union of South Africa.

McCann[73–76] has made use of supplementary nerve block with pentothal sodium anesthesia for general surgery. Relaxation for abdominal surgery was achieved by transincisional blocking of the intercostal nerves within the rectus sheath. He prevented deep reflex stimulation of respiration by anterior splanchnic and mesenteric block in intra-abdominal surgical procedures. Transincisional intercostal block was done during radical mastectomies. The quantity of pentothal necessary for any general surgical operation was reduced by almost 50 per cent when such supplementary nerve block was employed.

Ingraham et al.[77] found that local reduction in cutaneous temperature facilitated the introduction of needles through the skin of children for the purpose of medication or venipuncture. They used an insulated, ice-filled copper tube, which maintained a constant temperature for several hours. Application for a few seconds produced sufficient cooling for painless skin puncture.

Complications

Every method of effecting anesthesia for surgical procedures over an extended period of use has been accompanied by certain complications. Regional anesthesia, however, has its specific category of complications, chief among which are nerve paralyses and subarachnoid sepsis. Nicholson and Eversole[78] recently presented a review on the neurologic complications of spinal anesthesia. Steinberg and Bishop[79] and Fairclough[80] reviewed the pathogenesis of abducens-nerve palsy after spinal anesthesia, and Rose and Pritzker[81] reported a recent case. Evans[82] collected a number of cases of meningitis following spinal anesthesia and outlined a technic for prevention. Kennedy et al.[83] described 3 cases of fibrinous arachnoiditis after spinal anesthesia. Chivers[84] reported that the incidence of pneumothorax with intercostal nerve block was 19 per cent.

Replacement Therapy

That a real need exists for better methods of evaluating the effectiveness of modern preoperative preparation of the poor-risk surgical patient is keenly appreciated by both the surgeon and the anesthetist. Too often, a patient who is considered adequately prepared shows signs of shock in the operating room or develops a serious postoperative surgical complication. Recently, attention has been directed to the decreased blood volume in these chronically ill patients. Lyons et al.[85, 86] introduced the term "chronic shock" to describe this condition. The complete syndrome consists of weight loss, decreased blood volume, decreased blood protein and increased interstitial fluid volume. The authors showed that weight loss was correlated with reduced total mass of blood proteins without significant alteration of blood protein concentration. Lowering of the total hemoglobin mass, as in chronic bleeding, leads to the loss of plasma from the vascular bed to the interstitial fluid compartment. A reduction in plasma volume, as well as a loss of total hemoglobin, may mask deficiencies in the total mass of blood proteins. Ordinary methods of blood determinations, they contend, will then fail to show the reduction of blood volume and the attendant increased susceptibility to shock. Lyons et al. stated that corrective therapy consisted of whole-blood transfusion. In their studies adequate correction of chronic shock necessitated an average of 2700 cc. of blood in the preoperative period. After such treatment the plasma-volume increase approximates but rarely equals or exceeds the gain in erythrocyte volume. Consequently, the restoration of the hematocrit value to 50 per cent was advocated as an index of adequate therapy. A fundamental disturbance in hemoglobin metabolism was shown to be present in patients suffering from cancer.

Davidson and his associates[87] studied blood changes in patients with acute medical illnesses. They found that patients exhibiting peripheral vascular collapse had hyperglycemia, lactacidemia and a decrease in the bicarbonate reserve. There was also usually a lengthening of the prothrombin time and an elevation of the icteric index.

Scheinberg et al.[88] reported on homologous serum jaundice and warned against injudicious use of blood plasma from pooled lots.

Summary

The medical literature since World War II has introduced many new advances in regional anesthesia. Segmental spinal anesthesia and the addition of vasopressor drugs to agents used for spinal anesthesia are advocated as aids in decreasing the concentration and total amount of spinal drug necessary. Better control of the hypotension seen with spinal anesthesia is offered by new vasopressor drugs. The investigation of synthetic hypnotics gives promise of better preoperative and postoperative management of the surgical patient's apprehension and pain. Sympathetic-nerve block has proved an effective therapeutic agent in many disorders heretofore uncontrollable. The use of weighted spinal-anesthetic solutions for vaginal delivery offers a safer agent for obstetric anesthesia, and finally, the recognition of the serious depletion of whole blood volume in chronically ill patients throws new light on the preoperative preparation of this group.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 34191

PRESENTATION OF CASE

First admission. A twenty-seven-year-old housewife entered the hospital because of shortness of breath.

This symptom had bothered her as long as she could remember. She had always restricted her activities and found running impossible. She slept propped up on at least two pillows and had often been aware that the heart beat forcefully. Dyspnea and orthopnea became gradually severer during the two years before admission, and several new symptoms developed. These consisted of a dull ache to the left of the sternum, at first only after eating and then almost constantly, ankle swelling after standing for any length of time and cyanosis of the face, lips and fingernails aggravated by exertion. Two months before admission a new type of chest pain was noted, a constricting sensation beneath the sternum, associated with exertion, disappearing on rest and nonradiating. During the month before admission the patient was confined to bed with a respiratory infection. She entered the hospital because of failure to improve.

There was no history of rheumatic fever or other heart disease. Upper respiratory infections were frequent during the winter prior to admission, and the patient was bothered by a chronic, dry cough. There was no history of hemoptysis, night sweats or pleural pain. Seventeen years before admission she had entered the hospital because of polyuria and polydipsia. Physical examination was essentially negative. The chest was clear, and the heart sounds were unremarkable except that the pulmonic second sound was split and the same double sound was heard at the apex but to a less marked degree. No murmurs were heard. Examination of the blood was negative. The urine was not remarkable, except that the specific gravity seldom exceeded 1.002. A diagnosis of diabetes insipidus was made, and the patient was given surgical pituitrin, which controlled the symptoms. At home she continued the drug, two or three injections a day for fifteen years until two years before the present admission, when this treatment was stopped and the symptoms did not recur. The onset of catamenia had been very irregular. She had been married twice but had never become pregnant.

Physical examination showed an intensely cyanotic, rather obese woman lying propped up in bed. The veins of the fundi were tortuous and engorged, although the neck veins were not distended. The chest was clear to percussion and auscultation. The left border of cardiac dullness was at the midclavicular line; the right border was not percussible. The most forceful impulse was in the third and fourth left interspaces; it was much stronger than the apical beat. The pulmonic second sound was very loud, and the pulmonic first sound

moderately so, and the other heart sounds were distant. There was a soft, short diastolic murmur best heard in the third left interspace. The abdomen was obese and showed striae, and palpation revealed tenderness in the right lower quadrant. No organs or masses were felt. There were clubbing and cyanosis of the fingers and toes.

The temperature and pulse were normal. The respirations were 40. The blood pressure was 108 systolic, 80 diastolic.

The urine gave a +++ test for albumin and had a specific gravity of 1.006; the sediment contained epithelial cells. The red-cell count was 8,450,000, the hemoglobin 18.5 gm., and the white-cell count 9550. The fasting blood sugar, the nonprotein nitrogen, the carbon dioxide, the total protein, the sodium and the chloride were all within normal limits. The venous pressure was equivalent to 120 mm. of water; the circulation time was 8 seconds with ether and 30 seconds with decholyn. An x-ray film of the chest showed enlargement in the region of the left auricle and ventricle. The pulmonary artery and lung roots were prominent. An electrocardiogram revealed sinus rhythm, with a rate of 85, and a PR interval of 0.24 second, a QRS complex of 0.09 second, normal voltage, right-axis deviation, a slurred R wave in Lead 2, sagging ST segments in Leads 2 and 3, flat T waves in Leads 1 and 2, a very low T wave in Lead 3 and sagging ST segments in Leads CF_2, CF_4 and CF_5, with a prominent S wave in Lead CF_5 and inverted CF_2, CF_4 and CF_5. The patient improved on small venesections, digitalis and ammonium chloride. She was discharged on a low-sodium diet to be followed in the Cardiac Clinic.

Second admission (two months later). The patient did well on markedly limited activity until two months later, when she had an attack of breathlessness and substernal pain and was readmitted to the Emergency Ward. The physical findings had not changed. The diastolic murmur was described as a rumble, and an early diastolic blow was heard in the second left interspace. She improved on aminophyllin and was discharged after a two-day stay.

Third admission (five months later). During the period after discharge she was treated with venesection on several occasions, which lessened the symptoms. During the five days before re-entry she had three attacks of severe, stabbing pain in the left breast. Nausea, vomiting and great anxiety were also prominent symptoms.

Physical examination revealed an extremely agitated, cyanotic woman, lying flat on a stretcher. The pulse rate was 100, and the respirations 40. The blood pressure was 80 systolic, 60 diastolic. There was no venous distention with the patient sitting up. The chest was clear, and the heart unchanged. The liver edge was not felt. Five hours later the patient was examined by a consultant, who found in addition to the above findings a protodiastolic gallop and many dry, crepitant rales. Pressure on the right upper quadrant did not distend the cervical veins or reveal pulsations, but while this procedure was being done the patient suddenly stopped breathing, became pulseless and died.

Differential Diagnosis

Dr. Howard B. Sprague: May we see the x-ray films?

Dr. Stanley M. Wyman: The heart shadow is enlarged as described in the record. The left auricle appears to have displaced the left main-stem bronchus upward and to have narrowed it slightly. The enlargement of the left auricle is seen in the oblique view; I do not believe that I can say whether the enlargement of the ventricles is to the left or the right from the films alone. The heart comes far forward to the chest wall, strongly suggesting that the right ventricle might be large, and far posteriorly, which makes one question the left ventricle also. The main pulmonary artery is strikingly prominent in the anteroposterior projection, and the left and right main pulmonary arteries are more prominent than usual. The aorta is small. The lung fields are essentially clear. There is no evidence of pleural fluid.

Dr. Sprague: There is no pulmonary lesion that you can find, except vascular?

Dr. Wyman: I see no localized pulmonary disease. I cannot detect any intracardiac calcification on the films. I do not know about fluoroscopy.

Dr. Sprague: The right-axis deviation in the electrocardiogram is an important finding. It tends to contradict the observation of the left ventricular enlargement by x-ray examination.

I am going to start by saying that this is a case of pulmonary hypertension and see where we can go from there. The chronology is important. This woman had been dyspneic all her life, which is in favor of some type of congenital lesion, cardiac or pulmonary. At the age of ten years the examination showed no cyanosis or clubbing, no heart murmurs and no other cardiac abnormalities, except for a split second sound at the pulmonary area, and a negative blood examination. At the age of twenty-five dyspnea and orthopnea had increased and a dull ache developed at the left of the sternum, with ankle swelling on standing, cyanosis of the face, lips and fingernails, worse on exertion, and chronic dry cough. At twenty-seven years of age the patient entered the hospital, and examination showed obesity, intense cyanosis, a forceful impulse in the left third and fourth spaces, a loud pulmonic second sound, a soft diastolic murmur along the left upper sternal border and clubbing of the fingers and toes. Is there any note about the degree of clubbing?

DR. TRACY B. MALLORY: It was rather severe clubbing.

DR. SPRAGUE: The significant laboratory findings are a red-cell count of 8,500,000, with a hemoglobin of 18.5 gm., somewhat increased venous pressure, with normal arm-to-lung but prolonged arm-to-tongue circulation time. Of course this chronology rules out at once the congenital cardiac conditions associated with cyanosis and death in early life because this is primarily a patient with delayed cyanosis, and for that reason I have to assume that there was some change in the pressure relations in the circulation if we are going to invoke a veno-arterial shunt. There may previously have been an arteriovenous shunt, but whether or not we invoke this change in circulatory dynamics the evidence does point toward changes in the pulmonary circulation as being most important. If we assume that there was a congenital cardiac condition, what are the arteriovenous shunts, not associated with cyanosis in early life, that may become veno-arterial shunts if there is a change in the pressure relations? One should mention interauricular septal defect, with or without mitral stenosis, congenital or acquired — a Lutembacher syndrome; a high interventricular septal defect; patent ductus arteriosus; and, finally, Eisenmenger's complex. Regarding the pulmonary conditions, if we assume that we are dealing primarily with a pulmonary situation, with secondary cardiac changes of cor pulmonale, there may have been congenital anomaly, hypoplasia of the vascular tree or a change of some sort in the endothelium of the arterioles or capillaries. There may have been endarteritis or possibly secondary changes in the pulmonary circuit due to prolonged arteriovenous communication, as in some cases of patent ductus, which transposes the systemic pressure to the pulmonary side and results in changes in the lung vessels, causing delayed increase in pulmonary blood pressure. Finally, there are the unexplained changes in the lung of the Eisenmenger complex. In that condition there are a dextroposed aorta, a high interventricular septal defect and a large pulmonary artery as contrasted with the small pulmonary artery of the tetralogy of Fallot. In the Eisenmenger complex there is no obstruction in the outflow tract of the right side of the heart, but increased peripheral resistance in the lung, and little or no cyanosis, or delayed cyanosis. I think this development of heart symptoms with severe compensatory polycythemia and chest pain can be interpreted under the term of "angina hypercyanotica," which is described in conditions of high pulmonary pressure and polycythemia, although in this case I cannot rule out some degree of coronary insufficiency. Against interventricular septal defect, with or without mitral stenosis, clinically, is the fact that this woman was obese. These patients tend to be of slight build. There was not the striking increase in the pulmonary vascular markings typically associated with an interauricular septal defect, in which there is tremendous increase in the pulmonary flow. Furthermore, in interauricular septal defect the cyanosis — the "cyanose tardive" of the French — is not of long enough duration before death to be associated ordinarily with clubbing of the fingers. In interauricular septal defects one frequently sees auricular fibrillation, and, furthermore, there was no history in this case of rheumatic infection, which patients with interauricular septal defect are very prone to have.

As against uncomplicated high interventricular septal defect, I am worried about the absence of a systolic murmur because the condition is clinically associated with a loud systolic murmur and thrill. Against patent ductus arteriosus, again, is the absence of anything characteristic of the continuous murmur of patient ductus arteriosus. Against the Eisenmenger complex again there is no evidence of interventricular septal defect. There was no murmur, and although a soft early diastolic murmur had developed along the left sternal border, I am more willing to call that a functional murmur, a Graham–Steell murmur, rather than that of aortic regurgitation, which may be found with the Eisenmenger complex, because the aortic valve may be involved in the anomaly high up in the septum.

The evidence for primary pulmonary disease of the small arterioles is that the findings were those of dyspnea without diagnostic murmurs, and I might be inclined to think that we are dealing with a congenital vascular anomaly of the pulmonary bed. I believe that with such a condition the patient would not have gone so long as this without cyanosis and failure.

The lack of support for the diagnosis of veno-arterial shunt is the prolonged arm-to-tongue time. If something is injected into the vein and goes from the right side of the heart immediately over to the aorta, there should be shortening of the circulation time to the tongue. On the other hand, there may be such a slight venoarterial shunt that that is not good evidence. In this sort of situation Taussig[1] has recently emphasized the trial of inhalation of oxygen as being diagnostically valuable because if the cyanosis is due to a pulmonary condition, it may disappear. But in the Eisenmenger complex the oxygen saturation of the arterial blood will not be normal because there is still some degree of shunt of venous blood between the right ventricle and the aorta.

The events in the last seven months of the patient's history are attacks of breathlessness and substernal pain and what is described as a diastolic rumble. I do not know whether that has been added to make one think this was a Lutembacher syndrome with mitral stenosis, or whether it was an interpretation of one observer of what was later

called a gallop rhythm, perhaps gallop rhythm of the right ventricle. The final severe stabbing pain in the left breast, the fall in pressure, the absence of venous distention and the sudden death could have been due to either coronary-artery or pulmonary failure.

In summarizing this rather complicated situation, what do we have as possibilities? I should say primary pulmonary vascular anomaly that the patient had had all her life. There is no reason to consider that she represents the type of patient we have seen in these sessions in whom there has been pulmonary endarteritis or an obstruction of the finer vascular radicles. There are cases of multiple emboli, such as those described by Castleman and Bland.[2] The symptom of dyspnea in this case, however, was present all her life. Any arteriovenous shunt, I think, may well lead to greater pressure strain on the lesser circulation relative to that on the systemic. Volume strain is usually tolerated better than pressure strain on the pulmonary circuit. However, when these conditions exist more changes in the pulmonary vessels may ensue. These, in turn, increase the pulmonary pressure and result in reversal of an arteriovenous shunt, as in interauricular or interventricular septal defect, or even patent ductus arteriosus. That possibility has been advanced recently as another reason for ligation of patent ductus arteriosus.

I do not believe that we are dealing with idiopathic pulmonary fibrosis with cor pulmonale, but I confess that we have little evidence beyond the long history and delayed cyanosis to support a diagnosis of Eisenmenger's complex, mostly because of the absence of murmur. Yet we have certain cases of tetralogy of Fallot in which there is also an interventricular septal defect without a systolic murmur. I think that can be explained only by the fact that there may be only a small pressure gradient between the right and left ventricles, and murmurs are suppressed by increased blood viscosity from the polycythemia.

That brings us to what happens in the lung in the Eisenmenger complex. With a large pulmonary artery and an interventricular septal defect one would expect that the blood from the right side of the heart would readily pass into the pulmonary artery and not go into the left side of the heart and aorta to cause cyanosis. That is what happens in early life, but for some reason this increase in peripheral resistance in the lung occurs and cyanosis develops, mainly because of faulty oxygenation in the lung and also because of venous admixture through the ventricular septal defect. I am therefore going to vote for a combination consisting of a dextroposition of the aorta, explaining perhaps the small aortic shadow, the large pulmonary artery and a high interventricular septal defect. Thrombosis probably occurred in the pulmonary vessels. I cannot say just what may be found anatomically in these vessels, but there will be some type of abnormality. The patient probably had some coronary insufficiency, either from anoxemia and increased blood viscosity, in addition to what was a real anemia after the venesections, or from functional pulmonary-valve regurgitation, which the pathologist will probably not be able to prove.

DR. MALLORY: I take it you are not interested in the diabetes insipidus?

DR. SPRAGUE: It did bother me, but I cannot fit it in except as a manifestation of another congenital anomaly, although we have no history of any familial incidence.

DR. JOSEPH AUB: I am interested by the fact that when the patient was ten years old there was no suggestion of cyanosis.

DR. SPRAGUE: She had an increased pulmonary second sound and was unable to breathe comfortably.

DR. AUB: The same thing that was bothering the pulmonary vessels may have bothered the pituitary vessels when she was ten. How is that?

DR. SPRAGUE: Good.

DR. AUB: Then the whole thing would have been vascular.

DR. SPRAGUE: She pulled through the diabetes insipidus.

DR. AUB: She had extensive circulation.

A PHYSICIAN: How do you explain the increased PR interval?

DR. SPRAGUE: I think that the increased PR interval was nonspecific.

CLINICAL DIAGNOSES

Congenital heart disease, incompletely diagnosed.
Pulmonary endarteritis?

DR. SPRAGUE'S DIAGNOSES

Interventricular septal defect.
Dextroposition of aorta.
Thrombosis of pulmonary vessels, probable.

ANATOMICAL DIAGNOSES

Pulmonary fibrosis and emphysema, with slight bronchiectasis.
Cor pulmonale.
Hydrothorax, bilateral.
Chronic passive congestion.
Pulmonary arteriosclerosis, slight.
Hyperplasia of bone marrow.
Adenoma of thyroid gland.

PATHOLOGICAL DISCUSSION

DR. MALLORY: I have to present my evidence visually — otherwise you will not believe me. We found a marked cor pulmonale. The heart weighed 450 gm. The right ventricle was almost as large as the left, measuring 12 mm. in thickness. There were no congenital abnormalities of any sort.

The lungs, in contrast, showed a diffuse fibrosis and emphysema (Fig. 1). Here is a focal granuloma in the lung with numerous foreign-body giant cells, faintly suggestive of sarcoid (Fig. 2). Here is an area of bronchiectasis surrounded by rather extensive bands of fibrosis. I think this case is one of true cor pulmonale. What the explanation of the pulmonary changes is remains very much more of a problem. The patient was very young to have a severe emphysema. When we do see it in young people it is usually associated with evidence of some destructive fibrotic process that has occurred in the past. One group of such cases that we have recently been interested in here shows multiple sarcoid-like lesions throughout the lung. The evidence for that is minimal in the case under discussion. There were a small number of lesions such as the one I showed that suggested it, but if there ever was an extensive process of that sort, it had almost completely healed. It is also rather mysterious that there was no x-ray evidence of extensive pulmonary changes. The pulmonary vessels showed a few patches of atheroma but no narrowing of the vascular lumens. The bone marrow showed red-cell hyperplasia due to anoxemia, and there was a small coincidental adenoma of the thyroid gland.

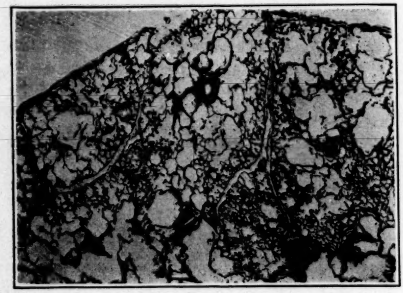

FIGURE 1.

DR. SPRAGUE: I have seen that and have been impressed with what we call idiopathic pulmonary fibrosis and emphysema, with absolutely normal chest films. It may be that the changes in the lung were on a congenital basis.

DR. MALLORY: No; I think they were acquired.

DR. SPRAGUE: As long ago as the patient could remember?

DR. MALLORY: Yes.

DR. AUB: Then how about the pituitary body?

DR. MALLORY: It showed extensive atrophy of the posterior lobe, quite characteristic of what is seen in diabetes insipidus. Why she should recover I cannot understand, because she had almost no posterior lobe left.

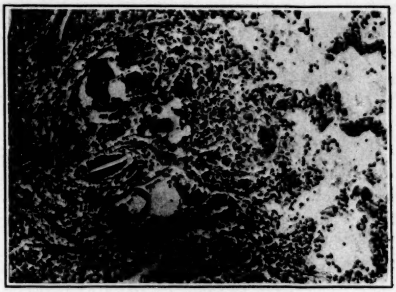

FIGURE 2.

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CASE 34192

PRESENTATION OF CASE

A fifty-two-year-old nulliparous housewife entered the hospital because of weakness and fatigability, pain in the left arm and leg, tinnitus, deafness and difficulty in swallowing and talking.

Fatigue and dyspnea were first noted two years before entry and became progressively worse. For several months the patient was able to perform only a minimal amount of housework. During the same period she had almost continuous severe steady pain in the left arm and "over the breast" associated with a sensation of coldness. Four months before admission there was a rapid change in the character and severity of the pain. The entire left side of the body became involved in an agonizing pain, constantly running up and down the left side of the body. Coincident with this

change in the pain there was a constant ringing in both ears, and three months later hearing became impaired. The tinnitus gradually lessened and on admission was entirely absent. There had not been any vertigo or nausea. During the few weeks before admission, while in another hospital, the patient developed a rapidly progressive weakness of the right facial nerve and difficulty in swallowing and talking. While there she required tube and intravenous feedings.

Physical examination revealed a thin, debilitated and dehydrated woman, who was alert and oriented. The heart, lungs and abdomen were normal. All four extremities could be moved equally, though feebly, and she could walk with assistance. The fundi were normal. The right pupil was 3 mm. and the left 2 mm. in diameter; both reacted to light. There was no nystagmus. There was palsy of the left external rectus and superior oblique muscles. The left palpebral fissure was narrower than the right. There were bilateral paralysis of masseter and pterygoid muscles and sensory loss in the left facial nerve, with a diminished corneal reflex and conjunctivitis of the left eye. Motion in the right lower part of the face was absent, and on the left was limited to a feeble upward twitch of the corner of the mouth. The sense of taste was lost. There was gross diminution of hearing bilaterally. Bone conduction was greater than air conduction. The Weber test was negative. The gag reflex was absent, and the patient was unable to swallow. There were marked weakness and slight atrophy of the tongue. The deep tendon reflexes of the arms were hyperactive, more so on the left. They were absent in the legs. The abdominal reflexes were absent, and the plantar reflexes were normal. Stereognosis and vibration sense were normal bilaterally.

The temperature was 98°F., the pulse 90 (equal and synchronous), and the respirations 25. The blood pressure was 178 systolic, 110 diastolic.

Examination of the blood at the other hospital just before admission disclosed a red-cell count of 5,000,000, with a hemoglobin of 13.2 gm., and a white-cell count of 6400, with 68 per cent neutrophils. The urine was normal. The blood sugar was 130 mg., and the nonprotein nitrogen 33 mg. per 100 cc. The sedimentation rate was 85 mm. per hour. A lumbar puncture done three weeks before entry showed an initial pressure equivalent to 200 mm. of water, which rose to 240 mm. on jugular compression and fell to 120 mm. after removal of slightly xanthochromic fluid, which contained 5 lymphocytes per cubic millimeter and gave a total protein of 250 mg. per 100 cc. with a +++ Pandy test. Ten days later the initial spinal-fluid pressure was equivalent to 290 mm. of water, with a protein of 500 mg. per 100 cc., a ++++ Pandy test and 18 lymphocytes per cubic millimeter.

On the first night the patient became very noisy and apprehensive and was given paraldehyde by rectum. A few hours later the respirations suddenly fell to 6 per minute. Oxygen was given by a Boothby mask. The temperature remained normal. The patient died in respiratory failure twenty-four hours after admission.

Differential Diagnosis

Dr. Roy L. Swank*: Stereognosis and vibration sense were normal bilaterally, but we are not told about other sensations. Should we assume that they were normal?

Dr. Charles S. Kubik: The sensations of touch and pinprick were said to be normal.

Dr. Swank: I believe that the patient had a lesion in the brain stem or the cranial nerves emanating from it, or both, from the level of the fourth or fifth to the twelfth cranial nerve. This was certainly not a typical acoustic neuroma, although it may have been very atypical or bilateral. There are certain features that, in my mind, rule out these diagnoses: the absence of nystagmus, the absence of history of staggering or ataxia, the absence of papilledema and the order in which the symptoms developed. I also feel the same about other single tumors outside the medulla and pons in the posterior fossa. I am unable to make such a diagnosis.

Other extramedullary conditions are suggested by the fact that the patient experienced vertigo and dyspnea two years before entry, by the findings relative to the ear and by the sedimentation rate of 85 mm. per hour. The white-cell count was normal, however, and there was no significant increase in neutrophils. I wonder if we are dealing with a chronic basilar meningitis. Tuberculosis would have to be thought of. Could it have been present for two years? There is no history of cough, and no report of x-ray studies. It seems unlikely that active tuberculosis was present so long. There is no history of treatment with streptomycin, which might have altered the story. I am inclined to

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discard this diagnosis. Was an x-ray film taken of the chest?

DR. KUBIK: X-ray studies were not done in this hospital.

DR. SWANK: Chronic, nonspecific, basilary meningitis associated with chronic middle-ear disease is another possibility. One would have to assume that the middle-ear disease was bilateral. I consider this a difficult diagnosis to arrive at from the information at hand.

This brings me to two diagnoses, which I shall consider seriously. The first is a glioma of the pons. This is a slowly progressive condition involving the cranial nerves late and producing increase in cranial pressure late, if at all. I believe that this diagnosis is possible. The few cases of this condition that I have seen have had marked ataxia and nystagmus. I see no reason why this condition might not pick out other systems in the brain stem, however. A glioma of the pons must extend from the lower end of the medulla to the upper pons and probably the lower midportion of the brain on the left. There are several things that are difficult to explain on this basis. It leaves me unable to account for the findings relative to the ear. Bone conduction was greater than air conduction. To explain this I assume that the patient was grossly deaf and was not hearing the tuning fork, but feeling it. Also, tinnitus of the prominence described in this case is unusual in intramedullary lesions. The spinal-fluid protein was markedly elevated, much more than it usually is in glioma of the pons; I shall assume that a block had occurred late in the disease and that the elevated protein content was a result of the block. Had the puncture been done some months earlier, normal protein might have been present.

Lastly, I think it is necessary to consider a malignant tumor with metastases. Metastases to the dura and to the sheaths of the cranial nerves could produce the clinical picture under discussion. Symptoms due to this lesion may be difficult to understand even after post-mortem examination. If the history is correct, one must assume that the metastases were present for two years and also that the primary site, most likely the lungs, was overlooked. It seems to me that this history was very long for dural metastases. I would consider metastatic carcinoma an attractive diagnosis but not possible to arrive at under the circumstances.

As a most likely diagnosis in this case I have to fall back on tumor within the pons and medulla, a glioma. I am not happy about it, but I believe that it has the fewest objectionable features.

DR. AUGUSTUS ROSE: I am interested in the severe pain on the left side of the body, and I wonder if Dr. Swank would assume that that was "thalamic" pain.

DR. SWANK: That has been seriously considered because it is difficult to explain the pain on the basis of a lesion in the medulla or pons. If one assumes that it was thalamic, one also assumes that there were two separate lesions and that the third-nerve nucleus was missed. I believe that this is unlikely. The smaller pupil and the drooped lid might indicate third-nerve involvement. I consider it more likely that this was due to a lesion within the brain stem producing a mild Horner's syndrome. Conditions such as syringomyelia are apt to cause pain within the spinal cord, and perhaps a similar mechanism operated in the brain stem in this case.

CLINICAL DIAGNOSIS

Amyotrophic lateral sclerosis.

DR. SWANK'S DIAGNOSIS

Glioma of pons and medulla.

ANATOMICAL DIAGNOSIS

Metastatic adenocarcinoma, primary site undiscovered.

PATHOLOGICAL DISCUSSION

DR. KUBIK: I should like to say that this is probably one of the most difficult neurologic cases that has been presented here. Dr. Swank came very close to the diagnosis when he suggested the possibility of metastases. There was an unusual type of metastasis, consisting of carcinomatous invasion of the subarachnoid space with involvement of the cranial and spinal nerves. The metastatic tumor was made up of columnar cells, which formed perfect tubules and also spread out in a single layer, covering the inner surface of the arachnoid membrane, blood vessels and cranial and spinal nerves in a most unusual and fascinating way. There was invasion of the cranial and spinal nerves and slight subpial invasion of the cord and medulla. In spite of a complete autopsy no primary tumor was found. I cannot believe, however, that a neoplasm of this type could have been primary in the central nervous system, and I assume that the primary lesion was so small that it was overlooked. We have had two or three other cases of metastatic carcinomatosis of the subarachnoid space, though none with such extensive paralyses as this. In at least one case there were cells in the cerebrospinal fluid, and a clinical diagnosis of tuberculous meningitis had been made.

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THE JOURNAL does not hold itself responsible for statements made by any contributor.

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OFT IN THE STILLY NIGHT —

THE public, according to complaints received by the American Medical Association, believes that it is not getting the service it requires from its doctors. A particular grievance is the difficulty — on occasions the impossibility — of obtaining emergency medical service at night.

For various reasons a reactionary attitude toward self-sacrifice has set in since the war. The devotion to a common cause in those soul-trying years elevated many persons to a heroic state of mind. Now, at least in this country, "we the people" are demanding the pay-off. We want things material, and we want them now.

The medical profession must bear its share of this failure to see eye to eye and clearly. In too many phases of its work there has been an increasing divergence from the public interest rather than an improved cordiality in public relations. The trend toward socialization has been a trend also toward the impersonalization of medical service.

During the war, 60 per cent of the nation's physicians — and they comprised the older group — remained at home, assuming their absent colleagues' duties. They were, on the whole, overworked, but, on the whole, as old-timers they accepted the responsibilities that appeared to them to be part of the obligations of a career of service. Many of them now believe that others should bear the extra burden, and this is what the others seem unwilling to undertake. The youngsters seem to have failed to acquire the idea that they have accepted a calling.

If an obligation implicit in the practice of medicine now fails of spontaneous fulfillment, however, some organized measures must be taken to provide what is lacking. The American Medical Association suggests that county medical societies or urban groups maintain telephone exchanges that will accept the responsibility for locating physicians available for emergency calls.

The majority of the older practitioners have led lives so organized that they were never, year after year, "off call" without a substitute constantly available. It is one of the responsibilities that those who care for the sick have accepted and must continue to accept: those for whom they care must never be abandoned.

CRUSADE FOR CHILDREN

UNDER the banner of the Crusade for Children the United Nations Appeal for Children is conducting a campaign for voluntary contributions to relieve the suffering of starving children overseas. Of the national goal of $60,000,000, Massachusetts has been assigned a quota of $2,500,000, with $1,500,000 to be raised in the Greater Boston area. The funds will be distributed to twenty-six religious, labor and special relief groups to supplement the inadequate amounts that governments are presently able to appropriate for relief. These agencies include the In-

ternational Children's Emergency Fund, previously separately engaged in collecting money, which has now joined them in the united appeal.

The Crusade for Children is more than another campaign to persuade hard-pressed Americans to dig into their pockets for a "worthy cause." The appeal focuses attention on the plight of the children of Europe, too often overlooked in the dramatic and uncertain political situation. These children are in acute need, and quick and generous action is required to prevent the threatened disease, mental deterioration and actual death from starvation. An example of one phase of the desperate condition of European children is provided by a recent statement from the International Children's Emergency Fund, which pointed out that hundreds of thousands of infants and young children are getting no milk and that others, more fortunate, are limited to less than half a pint daily.* The Fund has been engaged in feeding nearly 4,000,000 children in twelve European countries, and extension of the aid to Germany and China is under consideration; in co-operation with the World Health Organization and the Danish Red Cross and its Scandinavian associates, the Fund is prepared to assist governments in mass-vaccination programs to check the spread of tuberculosis among the children of Europe. To these endeavors the Crusade for Children has now added the goal of saving 230,000,000 children from starvation and death.

In 1212 A.D. the children of Europe set out on the disastrous Children's Crusade to recover the Holy Sepulcher from the infidel. It is fitting that the present campaign should take the form of a crusade for children: reversing the unhappy sequel to that crusade of long ago, Americans can help give enough to keep the children of Europe alive and to give them the chance of growing up healthy in mind and body. A generous contribution today may save tomorrow's world.

*Release from United Nations International Children's Emergency Fund, dated March 2, 1948.

"YOUR WITNESS"

A DOCTOR'S failure to give his patient at least the degree of care and skill that is ordinarily used in his community is malpractice in the eyes of the law, and civil law requires that a patient be recompensed for the actual damage suffered from such malpractice. Whoever professes to see anything unfair about that should be thankful that he is not living in the time of Hammurabi (about [2100] B.C.), when the law required decapitation of the doctor whose patient's illness failed to respond favorably to treatment. How bitterly must the Babylonian patient of that day have regretted his doctor's very natural reluctance to undertake a cure, and with what alarm must he have viewed the increasing shortage of physicians! Forty centuries have improved conditions, but not to the point where all criticism has disappeared.

The press, the public and the legal profession point to what they consider a seriously weak spot in the doctor's reputation for unselfish public service. They say that doctors will not testify in court "against each other." They even say that it is hard to get doctors to testify "for each other." In fact, doctors do not like to go to court at all. As a result, many malpractice suits are said to be decided by juries whose only expert testimony comes from medical crackpots and disreputable hired quacks. If any or all of this is true it must be corrected.

It should at once be admitted that most doctors are reluctant to go to court, and the validity of some of their reasons should be examined. Doctors like to think of their time as more valuable than that of other people, but courts go out of their way to save a busy doctor's time. Doctors dislike the risk of being made monkeys of by belligerent attorneys, but no witness who avoids bumptiousness and confines himself to giving honest answers need fear a hostile attorney. Nobody likes the idea of testifying against his friends, but that is not what an expert witness is required to do. His obligation is to testify to the honest truth and let the jury worry about who gets hit.

And there are two sides to it. Court procedure, even now, retains some flavor of the medieval trials by battle in which plaintiff and defendant were represented by armed champions who slugged it out with more enthusiasm for winning than for establishing the truth of a claim. As expert witnesses physicians might feel easier in the kindly hands of the judge than under the practiced battle-

axes of the contending attorneys. No perfection is claimed for medicine, and there is no reason to believe that the law is perfect. Nevertheless, it would be extremely difficult to conceive of any law that would enable juries to decide medical cases fairly without the benefit of honest medical testimony and plenty of it.

This is no private matter that can be shrugged off. Doctors are news, particularly now when the right of organized medicine to manage its own affairs is being publicly questioned on economic grounds. This is no time for doctors to let the public say that it is being defrauded of its civil rights by a polite conspiracy of silence in the medical profession. Some modern Hammurabi might believe it and get the urge to dust off one of those old laws. Any thoughtful doctor would prefer to settle for an occasional morning in court.

MOSELEY PROFESSOR OF SURGERY

Dr. Francis D. Moore, of Boston, has been appointed Moseley Professor of Surgery at Harvard Medical School and surgeon-in-chief of the Peter Bent Brigham Hospital.

Dr. Moore, who received his medical degree from Harvard Medical School in 1939, has been assistant professor of surgery at Harvard Medical School, assistant surgeon at the Massachusetts General Hospital, consulting surgeon at the Massachusetts Eye and Ear Infirmary and an associate member of the staff of the Palmer Memorial Hospital. He has become widely known for his work in gastrointestinal and thyroid surgery, as well as for his studies with the Office of Scientific Research and Development on the care and treatment of patients with burns. His investigative activities have also included the application of radioactive isotopes to surgical problems. He is a member of the editorial board of the *New England Journal of Medicine*.

The selection of such a young man (the youngest ever named surgeon-in-chief at the Peter Bent Brigham Hospital) for these posts constitutes a great honor and one that is richly deserved. As a successor to John Collins Warren, Maurice Howe Richardson, Harvey Cushing and Elliott Carr Cutler, Dr. Moore will hold an enviable academic position in American surgery, and his status at the Peter Bent Brigham Hospital is one of equal eminence. Those who are familiar with his past achievements are confident that his service will continue the great traditions of his distinguished predecessors. Both Dr. Moore and the institutions concerned are to be congratulated on this choice.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

Darling — Charles B. Darling, M.D., of Waban, died on April 19. He was in his seventy-ninth year.

Dr. Darling received his degree from Harvard Medical School in 1894. He served in the United States Army Medical Corps in World War I and was a member of the original staff of the Free Hospital for Women, Brookline.

His widow, two sons and a grand-daughter survive.

McIntire — Herbert B. McIntire, M.D., of Cambridge, died on April 21. He was in his ninety-first year.

Dr. McIntire received his degree from New York University Medical College in 1882.

His widow and a daughter survive.

Robbins — Albert I. Robbins, M.D., of New Haven, Connecticut, died on April 14. He was in his thirty-fourth year.

Dr. Robbins received his degree from University of Vermont College of Medicine in 1939. During World War II he served with the United States Army in the Pacific area.

His widow, three sisters and four brothers survive.

MISCELLANY

HOSPITALIZATION OF VETERANS

The hospitalization of veterans by the Veterans Administration reached an all-time high of 109,325 on March 1, 1948. This number represents a gain of 1,874 over the previous peak of 107,451 on February 1, and an increase of 37,612 since July, 1945.

The number of veterans awaiting admission has also increased, largely owing to applications from veterans with nonservice-connected disabilities. The Veterans Administration is required to care for veterans with service-connected disabilities and may care for those with nonservice-connected disabilities if beds are available and if they affirm that they cannot afford to pay for treatment elsewhere.

CORRESPONDENCE

IN DEFENSE OF WHITE CROSS

To the Editor: In the February 12 issue of the *Journal*, in an article entitled "Principles of the Massachusetts Medical Society," former President Elmer S. Bagnall states:

> I remember that in that same committee [Committee of Public Relations] discussion of medical-care plans later developed and that, under the brilliant and aggressive leadership of Dr. James C. McCann, the Blue Shield was conceived and launched. At about the same time the White Cross was born. This was an experiment in medical care sponsored by men who were impatient with such "inadequate" concepts for solution of problems in medical-care distribution as the Blue Shield then seemed to them. I have always thought that these men could have performed a service by reporting on the causes of failure factually and objectively.

As this is the first request from a representative of the Massachusetts Medical Society for information about the White

Cross, it seems appropriate to supply the information, to correct some false impressions about the White Cross that are reflected in this quotation from Dr. Bagnall's article.

First, the White Cross was conceived and born in 1939 while one of the undersigned was president of the Massachusetts Medical Society. Dr. McCann was appointed chairman of the committee to conceive the Blue Shield by a successor to the presidency. Thus it is obvious that the White Cross antedated the Blue Shield and was not conceived in impatience or competition with the Blue Shield.

The facts concerning termination of the White Cross may be stated briefly. Early in the winter of 1942 the officers of Medical and Surgical Associates, who had assumed the responsibility of providing medical care to the subscribers of the White Cross realized that the majority of the participating family physicians and many of the consultants might enter the armed forces. Medical and Surgical Associates would then be unable to fulfill their obligations to the steadily increasing subscribers to the White Cross. Meetings were therefore held between the officers of the White Cross and Medical and Surgical Associates to meet this contingency. After a month of consultations it was decided that the possibility would actually become a fact, since the majority of participating physicians were among the younger active members of the profession. It was therefore decided that Medical and Surgical Associates should avail themselves of the opportunity under the agreement with the White Cross to terminate the contract for medical service on three months' notice. When this decision was announced the subscribers asked that it be reconsidered. Several more meetings were held at which the subscribers sought means of continuing the service during the war. At these meetings the enthusiasm of the subscribers for the service was ample evidence of their satisfaction with it. The subscribers formed an independent organization and begged the Medical and Surgical Associates to continue to take care of the existing subscribers even if the rates should be considerably increased. They said they wished this because they were receiving better medical care than they had received before. With the demands of the war, however, no solution could be found.

We consider it fair to state that the majority of the participating physicians were interested in this experiment in the provision of complete medical care on a voluntary prepayment basis. Though they had only received approximately thirty cents on the dollar for service rendered, they appreciated that more was not to be expected until the scheme had been in operation for several years. In the meantime the burden on any one physician would not be great.

Probably the most important factor in the slow though steady growth in subscribers was the opposition of many members of the medical profession to the service. Though this did not interfere with physicians' willingness to care for patients, it did retard group enrollment, so essential for rapid growth and a good distribution of medical risk.

We also wish to record the interest, time and effort that the lay board of the White Cross contributed to the organization and operation of this pilot study. It is interesting that the definitions of specialist and consultant devised for this service have since been accepted and used rather widely. The concept that medical service should be built about the family practitioner supported by adequate consultant and hospital services was satisfactorily applied. In retrospect it seems, however, that more organization of the consultant and hospital services is desirable in the interest of quality and economy.

This statement provides the objective thoughts desired by Dr. Bagnall. The White Cross was a war casualty. To insinuate any other hidden cause for termination of the contract by Medical and Surgical Associates is neither objective nor factual.

For the White Cross:
EDWARD A. TAFT, *vice-president*
HAROLD S. FULLER, *treasurer*
For the Subscribers Group:
HAROLD PUTNAM, *president*
For Medical and Surgical Associates:
ALLAN M. BUTLER, M.D.
ROBERT L. DENORMANDIE, M.D.
CHANNING FROTHINGHAM, M.D.
EDWARD L. YOUNG, M.D.

BOOK REVIEWS

Calcific Disease of the Aortic Valve. By Howard T. Karsner, M.D., and Simon Koletsky, M.D. 8°, cloth, 107 pp. Philadelphia: J. B. Lippincott Company, 1947. $5.00.

This volume, which is essentially a statistical monograph, analyzes the history and pattern of a condition that has borne various names but for which the authors consider the best to be "calcific disease of the aortic valve." To the complete material derived from this review of the literature they have added the detailed study of 200 cases observed at autopsy. The book serves, therefore, as a valuable reference work in which both the clinical and the pathological aspects of this disease are submitted to rigorous statistical criticism.

It is pointed out that Mönckeberg first studied the condition intensively and that the most important contributions to its understanding have followed his pioneer effort, even though his emphasis on the arteriosclerotic etiology of the valvular deformity has been supplanted by growing evidence that the cause is almost always rheumatic infection.

The authors consider the factors of age, sex and race, pointing out that calcific disease of the aortic valve is largely a disease of later life, that its preponderance in males is less than has been thought (1.6:1.0) and that it is infrequent in the Negro. The heart is generally large, in fact almost as large as that in syphilitic aortic insufficiency.

The pathological findings, with excellent illustrations, are presented in detail, as is the correlation of the aortic lesion with the stigmata of rheumatic involvement elsewhere in the heart.

The clinical examination is also carefully analyzed with consideration of angina pectoris, syncope, character of the pulse, murmurs, thrill, blood pressure and electrocardiogram. In only about half the cases in their series in which electrocardiograms were recorded was there left-axis deviation. The value of visualization of the calcified aortic valve area by x-ray examination is emphasized.

Sudden and unexpected death occurred fairly often, but most patients died of congestive failure.

Of 190 patients in whom a clinical diagnosis might have been made it was offered in only 48. Attention to the information and suggestions in this book should improve this diagnostic figure.

Nutritional Disorders of the Nervous System. By John D. Spillane, M.D. (Wales), M.R.C.P. (Lond.). With a foreword by George Riddoch, M.D., F.R.C.P. 8°, cloth, 280 pp., with 103 illustrations. Baltimore: The Williams and Wilkins Company, 1947. $5.00.

During World War II, owing to the long confinement of prisoners of war and persons in the concentration camps, there was an unusual chance to investigate nutritional disorders of the nervous system. The author studied patients with this type of disorder, particularly English prisoners of war in the Far East and civilians confined in Hong Kong. In addition to his personal observations he reviewed the reports of other physicians with similar experiences in the Philippines and elsewhere. Detailed case histories are given, with excellent illustrations both by photographs and by diagrams. The book contains references to the pertinent literature. This study is an outstanding one, far exceeding anything of its kind previously published.

Handbook of Psychiatry. By Winfred Overholser, M.D., Sc.D., and Winifred V. Richmond, A.M., Ph.D. 8°, cloth, 252 pp. Philadelphia: J. B. Lippincott Company, 1947. $4.00.

The authors have succeeded in setting forth modern psychiatric concepts in simple, nontechnical language. The volume has been prepared for intelligent lay persons and for relatives of mental patients, but it will probably be in the library of most specialists in psychiatry.

The material in the text, which has been presented in an undramatic, conservative manner, is easily read and well organized and written, with the exception of a few typographic errors. It may fail to reach the general public, as the authors hoped, because of its resemblance to a standard textbook of psychiatry.

Treatment of mental disorders is discussed only in a general way; self-treatment is discouraged. There is an excellent reading list at the end of each of the seventeen chapters.

The chapters on "alcohol and its role in the psychoses," "crime and mental disorder" and "psychiatric conditions in childhood," to mention only three, contain many facts and explanations that should be more generally known and understood by the public. The purpose of the authors to educate the average man and woman is indeed praiseworthy; the results may not be encouraging because the man on the street prefers picture magazines.

The clarity of the language is a tribute to the psychiatrist and the psychologist who collaborated in writing it. College students, nurses and other professional groups can read this handbook with ease and profit.

What is Psychology? A basic survey. By Werner Wolff, Ph.D. 8°, cloth, 410 pp., with 39 illustrations. New York: Grune and Stratton, 1947. $4.00.

The author of this volume hopes to arouse a healthy skepticism of the various theories and methods in psychology and to create an interchange among collegiate classes of experiments in "depth psychology." He would have done better with an epistemologic discourse or an experimental presentation, rather than the pseudoexperimental approach taken. It appears that too much ground has been covered in too short a space to do justice to the author's points of view. The result is somewhat of a hodgepodge of experimental material and hypothetical musings, which are used sometimes to substantiate and sometimes to refute the point at issue. As a text for use with the author's own classes it may serve its purpose; its value as provocative material in other areas is questionable.

Trichomonas Vaginalis and Trichomoniasis. By Ray E. Trussell, M.D. With an introduction by E. D. Plass, M.D. 8°, cloth, 277 pp., with 19 illustrations and 16 tables. Springfield, Illinois: Charles C Thomas, 1947. $6.00.

It was in 1836 that Donne discovered and named the protozoan parasite *Trichomonas vaginalis* but not until 1916 that Hoehne first described its pathogenic properties. During the past thirty years there has accumulated, on the subject of trichomoniasis and its treatment, an enormous literature that the present monograph aims to summarize and evaluate. The author has also done extensive biologic, experimental and laboratory research, and was the first to obtain the organism in pure culture. He describes in detail its history, morphology, chemical, cultural and staining characteristics, and discusses its pathogenicity and the clinical phenomena of Trichomonas infections. Finally he investigates its treatment and presents an alphabetical index and discussion of 185 chemical and other agents that have been utilized in efforts to eradicate it. It is noteworthy that, of the antibiotics, only tyrothricin and tyrocidin have shown any effect whatever.

The text of this authoritative work is adequately illustrated with 19 figures and 16 tables and has an exhaustive bibliography of 1586 references. One appendix summarizes the taxonomic relation of Trichomonas to other protozoa, and another presents a parasite-host list of 167 known species. There is an admirable introduction by Dr. E. D. Plass, professor of obstetrics and gynecology in the State University of Iowa, in whose laboratories and libraries the research was done.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Diseases of the Joints and Rheumatism. By Kenneth Stone, D.M. (Oxon.), M.R.C.P., physician, B.R.C.S. Clinic for Rheumatism and Physical Treatment, Kensington. 8°, cloth, 362 pp., with 58 illustrations. New York: Grune and Stratton, 1947. $6.50.

Fatigue and Impairment in Man. By S. Howard Bartley, Ph.D., professor of research in the visual sciences, Dartmouth Eye Institute, Dartmouth Medical School; and Eloise Chute, M.A., research associate in the visual sciences, Dartmouth Eye Institute, Dartmouth Medical School. With a foreword by A. C. Ivy, Ph.D., M.D., vice-president, Chicago Professional Colleges, University of Illinois. First edition. 8°, cloth, 429 pp. New York: McGraw-Hill Book Company, Incorporated, 1947. $5.50.

Unipolar Lead Electrocardiography: Including standard leads, unipolar extremity leads and multiple unipolar precordial leads. By Emanuel Goldberger, M.D., adjunct physician, Montefiore Hospital, New York City, cardiographer and associate physician, Lincoln Hospital, New York City, and clinical lecturer in medicine, Columbia University, Faculty of Medicine. 8°, cloth, 182 pp., with 88 illustrations. Philadelphia: Lea and Febiger, 1947. $4.00.

Radium Dosage: The Manchester system. Edited by W. J. Meredith, M.Sc., F.Inst. P., Christie Hospital and Holt Radium Institute, Manchester. 8°, cloth, 124 pp., with 38 illustrations, 4 plates and 27 tables. Baltimore: Williams and Wilkins Company, 1947. $4.50.

NOTICES

ANNOUNCEMENTS

Dr. Eugene Guralnick announces the opening of his office at 422 Beacon Street, Boston, for the practice of general surgery.

Drs. W. J. Mixter and C. G. Mixter announce the association of Dr. H. Thomas Ballantine, Jr., in the practice of neurological surgery at 319 Longwood Avenue, Boston.

MASSACHUSETTS MEDICO-LEGAL SOCIETY

The annual meeting of the Massachusetts Medico-Legal Society will be held at the Hotel Statler, Boston, on Wednesday, May 26, at 2:30 p.m.

PROGRAM

Business Meeting

The Ruxton Case — A Study in Scientific Criminal Investigation. Robert P. Brittain, M.A., B.L., LL.B., M.B., Ch.B., Glasgow, Scotland.

Refreshments

UNIVERSITY OF PENNSYLVANIA MEDICAL ALUMNI SOCIETY

The University of Pennsylvania Medical Alumni Society will hold a dinner at the Convention of the American Medical Association in Chicago on Wednesday, June 23, at the Lake Shore Club, 850 Lake Shore Drive. On arrival in Chicago, alumni should communicate with Miss Frances R. Houston, executive secretary of the Medical Alumni Society, at the University of Pennsylvania registration booth.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING

THURSDAY, MAY 13

FRIDAY, MAY 14

*9:00–10:00 a.m. Zinc Content of Whole Blood in Normal People and in Patients with Blood Dyscrasias. Dr. John G. Gibson, 2nd. Joseph H. Pratt Diagnostic Hospital.

*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

12:00 m.–1:00 p.m. Clinicopathological Conference (Boston Floating Hospital). Joseph H. Pratt Diagnostic Hospital.

TUESDAY, MAY 18

*12:00 m. X-Ray Conference. Margaret Jewett Hall, Mt. Auburn Hospital, Cambridge.

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

(*Notices concluded on page xvii*)